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CHRONIC INDIGESTION.

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THE complaint of indigestion is probably the commonest statement made by a patient seeking medical advice. In 200 consecutive private patients 70% made this complaint amongst others. So widespread in its manifestations is the group of illnesses covered by this term that one can do no more in a single evening than touch on certain aspects of the subject.

To the average layman the term "indigestion" is a very comprehensive one, covering a large number of diverse symptoms, having only this in common, that they are associated with, or are thought by the patient to be associated with, a derangement in the process of digestion.

These symptoms are commonly centred in the abdomen or the lower part of the thorax and can be briefly summarized as: (*a*) a sense of fullness after eating or, conversely, a feeling of unnatural emptiness, usually relieved by taking food; (*b*) an actual pain associated with food; (*c*) a soreness localized to the supposed surface sites of the digestive tract from the throat downwards; (*d*) belching of wind; (*e*) regurgitation of mouthfuls of food; (*f*) vomiting; together with more direct symptoms, such as cardiac palpitation, furred tongue, constipation *et cetera*.

It should not be necessary to remind a professional audience of the self-evident fact that indigestion is a symptom complex and not a disease in itself; yet the frequency with which the patient hopefully asks for a confirmation of his complaint as being "just indigestion" suggests that the education of the layman, at least in this matter, is still incomplete. The willingness of the patient to accept this term as a diagnosis may in part account for the incomplete investigation that is often accorded to the symptoms of chronic indigestion.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on February 2, 1934.

The factors producing indigestion are not necessarily abdominal, and disorders of practically every portion of the body may cause derangement of the digestive function. In particular, general metabolic disturbances, chronic sepsis, nervous and cardiac causes must be constantly remembered in this relation.

Although the actual stimulus to intestinal derangement may be of such a various nature, in so far as a dyspeptic symptom is actually gastric in production, the mechanism of its production is relatively simple. There is every reason to believe that the only stimulus that can act on the stomach and give rise to symptoms is one expressing itself in disordered motor function.

In particular, no variations in quantity and quality of secretion, no bathing of ulcerous or other lesions with ingested irritants is itself appreciated as discomfort, except through its effect on gastric motility.

The motor functions of the stomach can be briefly described as the maintenance of tonus and the propulsion of contents by caudad peristalsis, such peristalsis embodying a wave of relaxation followed by a wave of contraction. Should either tonus become greatly excessive or a defect in the peristaltic waves produce spasmodic contraction, a painful cramp is set up in the stomach. It is characteristic of such a cramp that it is relieved by any factors that force a change in the content of the stomach, such as vomiting on the one hand, or gas production by alkaline powders or gaseous drinks on the other. The pain so produced is diffuse in nature, roughly centred in the mid-line of the epigastrium, and, according to its position in the stomach musculature, is appreciated near the epigastrium or *vice versa*.

Diagnosis.

The means at our disposal in making a diagnosis involve the study of: (*a*) the general type of the patient (diathesis); (*b*) history of complaint; (*c*) physical signs, including: (i) routine clinical examination, (ii) laboratory investigation, especially test meal examination, examination of fæces for occult blood, the Wassermann test, and (iii) radiological investigation.

The Type of Patient.

During recent years an awakening of interest in the patient as a whole, as distinct from his local disease processes, has occurred. It is a self-evident fact that there are distinct physical types, that certain types are more subject to certain disease processes than others, and, moreover, that one type of patient will consistently react to a given stimulus in a different manner from another physical type. By a study of their varying reactions it has been found possible to obtain certain type groups.

To the physicians of Guy's Hospital, and particularly to Hurst and to Ryle, we are indebted for the study of the relationship existing between general physical types and their abdominal counterparts and the derangements of digestion to which these various types are subject. These physicians have shown that a broad distinction can be made between two types of individual to whom they have applied the term "hypersthenic diathesis" and "hyposthenic diathesis".

The hypersthenic type is most often seen in men. It is associated with a sturdy, broad physical build, an active frame of mind, and normally the possession of good health. The wide subcostal angle is associated with a short transverse or steerhorn stomach, and the general bodily activity is reflected in the hypertonic, rapidly emptying, digestive tract. This rapid emptying is associated with free gastric secretion, and the test meal shows a high content of free acid and the typical "climbing curve". If a stimulus anywhere in the body produces a derangement of their digestive function, it is manifested in these patients by the production of late food discomfort, that is, they complain of a feeling variously described as unnatural emptiness up to actual severe pain three or more hours after food, and this feeling is typically relieved by taking further food. This response occurs quite irrespective of the stimulus producing the response, and is related absolutely to the soil and not to the seed implanted therein.

At the opposite extreme from the hypersthenic group we have the hyposthenic class. Typically thin and often tall, a large proportion appear to be females. They have not the robust health of the first-named group and are often sufferers from minor disorders. Although never very well, yet they seem to live to a normal span of life. Although they are especially subject to digestive disorders, their dyspeptic responses differ from those of the hypersthenic type. Discomfort is felt shortly after taking food, being variously described as a fullness or a pain, and it is usually relieved by any factors that will temporarily alter the stomach content, such as the gas production of alkaline powders or of gaseous drinks.

Here again the response is typical of the type, quite irrespective of the actual stimulus of production.

The stomach of these patients reflects the long narrow build of this type, and tonus is defective and emptying slow. The free acid content tends to be low or completely absent.

The History.

Since we see that the time relationship between food and discomfort is associated with the patient's type and not with the disorder from which he suffers, we must next inquire what points in the patient's history can be used to determine the actual nature of his complaint.

First we must obtain a detailed account of the history of the dyspepsia.

The first point of inquiry that may help is how long he has suffered from this complaint. If the patient is still young and the history is said to extend back to childhood, it suggests: (*a*) some con-

genital abnormality of the intestinal tract; (*b*) some basic metabolic disturbane, such, for example, as a dyspepsia associated wi migraine or with ketosis; or (*c*) a nervous dyspepsia, in which case associated symptoms of an abnormal nervous system will be elicited. A story extending back eight to ten years is typicallygiven by the subject of chronic peptic ulcer when first seeking serious medical advice, the average ag of such a patient at this time being thirty-eight to orty years.

Several years' history is commonly given by the subject of cholecystitis or viscroptosis. A history extending over only a few weks or months must be regarded very seriously. [t may indicate the early stage of any of these ore chronic ailments or may be associated with incmplete resolution of some abdominal state, such a acute appendicitis; but in every person of midle age or over the presumption should be cance and no other diagnosis should be made till this an be excluded.

Now an important questionomes: Does he suffer continuously or does the dyspsia recur in attacks? and if the latter, is he comptely free during the intervals? If attacks occur, hat is the length of the attacks and of the remissns?

Chronic gastric and duoderl ulcers are the most typical causes of the dyspsia which occurs in attacks with free intervals. t least in their early years the dyspepsia of chron peptic ulcer occurs in comparatively short attacs separated by long intervals: thus it is typical f duodenal ulcer to recur every six to nine month in an attack lasting perhaps two weeks. Cold wther often seems to precipitate an attack, and my of these patients are completely free of symms in the summer months.

The history of short attacand long remission is so constant in duodenal uer that without its presence the diagnosis can arcely ever be sustained, however strong the asciated evidence may be.

A more rapid alternation ofttack and remission favours other chronic inflamntory origins, especially chronic cholecystitis. Igall-bladder disease alternations of attacks and mission often occur every one to two weeks, an the length of the attack may approach the leng of remission. The dyspepsia associated with diant sepsis, such as chronic tonsillitis or apical toh infection, belongs frequently to this class.

I cannot emphasize toc strgly the importance of complete remissions in the fferential diagnosis between physical and so-calleenervous dyspepsia; the regular alternation o attk and remission is absolutely diagnostic of plysic dyspepsia.

When one is faced with a ptic ulcer in which either partial obstructin bm scarring has occurred or in which the base othe ulcer is formed of extragastric tissue, sucl as te pancreas or liver, a constant dyspepsia ma be present. Here the earlier history and associate symptoms should help. Thus in the obstructive type pain will be experienced late in the ight, many hours after food is taken, or vomiting will occur, while in the posterior perforating type the probable association of back pain will assist.

A shorter constant history makes one suspect carcinoma.

The great majority of the patients in whom true remissions are absent belong to the two groups of nervous dyspepsia and right visceroptosis with hypotonicity. These two groups include the large majority of all cases seen in Brisbane and will be considered carefully later. It must suffice now to say that above all others they are groups in which a careful history is invaluable to the diagnosis.

The question of remission having been settled, we must next inquire into the exact nature of the discomforts experienced during an attack, and in particular (i) the type of discomfort and (ii) its distribution during the day.

The hypersthenic type is not often a nervous type; so the symptoms of this type, an empty feeling or a nagging or toothache-like pain appearing several hours after food, and relieved by food, is usually of physical origin.

The early symptoms of the hyposthenic type are more often of the nature of uncomfortable fullness than of actual pain; though the more direct the stimulus, as in the case of gastric ulcer, the more likely will the complaint be one of definite pain.

The site of the pain is important. Remember that all intrinsic gastric pain is centred on the mid-line, between the xiphisternum and about an inch above the umbilicus, the actual site varying with the height of the motility upset in the stomach.

The distribution of the discomfort throughout the day is of paramount importance. Is pain complained of before getting out of bed in the morning? Only a high grade obstruction can produce pain ten or twelve hours after food; therefore the complaint that the discomfort is as bad as ever on awakening suggests strongly that the complaint is not a dyspepsia.

Most physical causes proceed to operate a certain time after taking food, the actual time varying with the diathesis. The closer the stimulus, the more exactly appears the definite relationship.

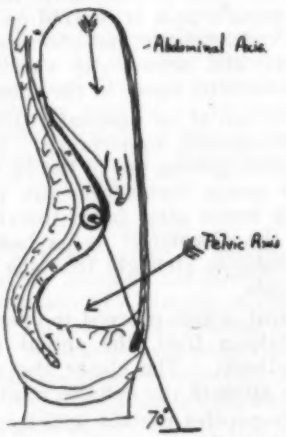
In some dyspeptics certain articles of food tend to produce discomfort more than others. Thus the subject of cholecystitis usually finds that certain fried and fatty foods, which provide a strong stimulus to biliary function, especially produce discomfort. This type of patient is to be differentiated from the neurotic dyspeptics, who act on the false theory that some article of food must be responsible for their indigestion and who discontinue taking one food after another till they are practically starving.

Another large group of patients are comfortable during the early part of the day and complain only in the latter part when they are beginning to get tired, or they complain on working days and are free on Sundays. This is the very important visceroptotic group.

The subject of right visceroptosis is so important, and yet so much the subject of ignorance and prejudice, partly at least through an indisposition to distinguish between the diagnosis of the disorder and its treatment by operation or otherwise, that I should like to make plain my views on the subject.

I hold that the primary fault in these patients is a lack of lateral fixation of the ascending colon. The caecum occupies a unique position in that it acts for hours at a time between meals as a reservoir for intestinal contents from which water is being extracted. In most animals the weight of this loaded caecum is supported by the anterior abdominal wall, but in those animals which have assumed the erect posture, including man, the lack of this support has made further fixation necessary, and in this group, but no others, the ascending colon has become fixed to the lateral abdominal wall by a peritoneal fold.

As Sir Colin MacKenzie has shown, fixation of the colon originates in the region of the spleen and progresses downwards both to the left and right. Fixation of the left colon has now become well established in the human being, but the right colon is still in the experimental stage, and more or less fixation is absent in about 20% of individuals, and it is amongst certain members of this latter group that symptoms occur.



It is interesting to observe the factors that tend to support a caecum in the absence of lateral fixation. These are: (i) Primitive dorsal mesentery. (ii) The obliquity of the pelvic brim, in so much as this normally maintains an angle of 70° with the horizontal. In a person with normal posture not only does a marked constriction exist between the abdominal and pelvic cavities, but also these cavities are acutely flexed on each other. The maintenance of this posture indirectly depends upon the maintenance of muscle tone. (iii) A flat abdominal wall. Here again muscle tone is of paramount importance. (iv) The relative weight of the caecum, and here it may be taken for granted that the more full the caecum is, the heavier it becomes. This factor of heavines depends again on efficient colonic function nd usually the presence or absence of constipation becomes the index of this function.

When these varios factors, in whole or in part, are defective, the cecum drops towards the cavity of the true pelvis, resenting added tension to the primitive mesentery and in these circumstances a sympathetic nervou irritation is set up, which in its turn causes cetain disorders of function in various portions of he intestinal tract and thereby produces symptoms

The factors favouing the production of symptoms in right visceroptosis are therefore, in order of importance, defectiv muscle tone, defective posture, and constipation.

As a matter of linical experience it has been noted on innumerale occasions that these factors are indeed so assoated; thus the right visceroptotic tends to get pai (i) as he or she becomes tired and the parietal nscles begin to relax; (ii) in certain positions here posture can exert an influence; and in tis relation heavy lifting in a bent posture standpreeminent, for in these circumstances not onlyare the abdominal and pelvic cavities merged in one straight tube, but an added muscular for raising intraabdominal pressure and pushing thcaecum from above is present.

Most visceroptoti can distinguish quite clearly that the better theibowels act, the less discomfort they suffer. Finall it need hardly be added that the symptoms of rit visceroptosis are essentially symptoms of the ect posture and are relieved usually entirely byecumbency.

Many patients tice the influence of posture when sitting slumpl in a chair. Pain coming on in these circumstaces can often be immediately relieved by straighting themselves up and arching their backs.

Before it can beaken for granted that a right visceroptosis is dictly related to the dyspepsia complained of, it often necessary to eliminate such factors as checystitis or ulcer, which many men think are comon sequelæ of absence of colonic fixation

In comparison wi other countries, and especially with those colder nds from which most of our medical periodical originate, a very distinct difference in the rative incidence of dyspepsia-producing factors n be noted here.

Chronic duodenaulcer is a relatively rare disease in Brisbane. Amongst 2,000 private dyspeptic patients only 4% ffered from this disease, and in most of these it vas relatively benign, yielding easily to medical nanagement.

In the twelve maths prior to June 30, 1933, only four deliberate oeratims for chronic duodenal ulcer appeared t have been performed at the Brisbane Hospital whil eleven emergency operations for duodenl perforation were performed during the same priod.

This differs very markedly from the English experiences. Ryle,[1] in his article on the "Natural History of Duodenal Ulcer", states that in his private practice, which has a general character with an abdominal bias, duodenal ulcer is his most frequent diagnosis, accounting for 5% of all cases. His ten most frequent diagnoses are: duodenal ulcer, anxiety state, constipation, cholecystitis and gall-stones, hyperpiesia, tuberculosis, anæmia, migraine, spastic colon and obesity.

In the practice of myself and my associates, which also has an abdominal bias, out of 7,000 patients only 77 were diagnosed as suffering from duodenal ulcer, although nearly 2,000 were classed as suffering from disorders of the intestinal tract.

The influence of climate on peptic ulcer has been incidentally observed before, as when Ryle says:

> I have had patients who were free from symptoms in India, but affected when at home, and Major E. B. Marsh, who made a particular study of duodenal ulcer among the troops at Aldershot, where he found it prevalent, later wrote to me from India to report that in twenty-two months in the East, among British troops, differing only that they were slightly older than the home troops, he had not seen a single proven case of ulcer.

In the same way recurrence of ulcer pains are most common in cold weather.

Low gastric acidity is relatively common in hot countries, and is certainly common in Brisbane, as has been shown by Nye and Sippe, among others; and this, together with the lowering of tone in striated and unstriated muscle that goes with hot climate, accounts reasonably for the rarity of the hypersthenic diseases as contrasted with the hyposthenic group.

Another group of cases shows a clear association with exertion apart from posture. This is the cardiac group, and in these patients the abdominal discomfort complained of may be actually an angina.

In these patients a relation to the filling of the stomach may be noticed. Thus a small meal may cause no distress, but a large meal will so distend the stomach that it seriously embarrasses the heart, with the production of pain. Other dyspeptic disturbances are the result of cardiac decompensation and nutritional upset, so resulting, in the abdomen.

The association of symptoms with nervous upsets creates a presumption of a nervous origin for the dyspepsia. By the term nervous dyspepsia I presume we refer to two groups of symptoms. In the first the symptoms are purely mental, originating in the mind and with a mental mechanism of the nature of a defence mechanism similar to other psychiatric symptoms. This class, I am sure, is rare. In the second we commonly mean, in simple language, that the stomach is being affected through the nerves. This is primarily a mental upset, of the nature of fear or anxiety or excitement, and this through the mechanism of the sympathetic nervous system or the internal secretions, especially that from the suprarenal medulla, causes certain motility changes in the stomach or intestines. Thus the actual mechanism of symptom production is identical with the method of production of symptoms from physical causes.

This association of the mind with intestinal function is of common observation, and the diarrhœa of fear or excitement or nervous tension is parallelled by the distaste for food or even vomiting that may occur under similar conditions. Thus Alvarez records that before an important speech Charles Darwin, a pronounced nervous dyspeptic, would vomit for a week. But note that the presence of nervous factors does not certainly imply a nervous cause. Nervous tension often aggravates a duodenal ulcer, while nearly anyone with peptic ulcer will get temporarily better if he goes for a holiday.

The constant association of dyspepsia with headache brings up the suspicion of migraine. The occurrence of attacks and the one-sided nature of the headache are important helps, though the complete relief after vomiting may suggest an abdominal cause, as, indeed, Crile believes often to be the case.

The association of constipation with dyspepsia is often observed. Usually both are symptoms of a common cause, such as right visceroptosis; and the improvement of one by the elimination of the other is merely an indication of the development of more normal function right through the intestinal tract.

In this relation one should remember that pain occurring soon after food is not necessarily of gastric origin. The intestinal tract is made up of a number of segments, and the passage of food into the upper one is a signal for a general move on in each segment. A disturbance of peristalsis in any segment may then become elevated to consciousness as a discomfort or pain, and the apparent relation to the intake of food may cause it to be erroneously related to the stomach rather than lower in the intestinal tract.

Inflammatory Attacks.

Many dyspeptics present in their history associated symptoms other than the true dyspepsia. In the first place it may be possible to elicit the story of a previous acute inflammatory attack, and in this way the subsequent or associated dyspepsia may be explained.

Thus an occasional patient will relate that some months ago, prior to the beginning of his dyspepsia, he was seized with an attack of umbilical pain, which, after some hours, settled down into the right iliac fossa, was associated with tenderness and finally almost subsided. An occasional discomfort is still probably felt in this site. In such a patient the diagnosis of incomplete resolution of an acute appendicitis may be made with great probability of correctness and any dyspepsia that has been present since this attack will be relieved by removal of the appendix.

More frequently an inflammatory attack settles the diagnosis in favour of a gall-bladder.

Thus in one patient, a bustling, successful business man, forty-two years of age, clearly of the hypersthenic type, a discomfort had been occurring some hours after meals and had been relieved by further food. Remissions occurred, but never longer than a month.

The consensus of opinion was veering toward chronic cholecystitis as the probable origin of his dyspepsia when, for the first time in his life, he suddenly developed an ultra-acute cholecystitis.

Some days later a gangrenous gall-bladder containing stones was removed with complete and permanent cure of the dyspepsia.

Just as acute pyelitis is notoriously associated with gastric upsets, and especially vomiting, so more chronic renal ailments are a frequent cause of dyspepsia. The history of urinary disturbances in the previous history should put one on guard against missing the diagnosis.

Recurring sore throats are a reminder that tonsillar sepsis is a potent cause of dyspepsia.

Some two years ago I had a patient referred to me whose dyspepsia of the late food type had continued unabated since a gastro-jejunostomy was performed, the excuse for which had been a radiological report of duodenal ulcer, and a very doubtful scar on the duodenal wall. Gross tonsillar sepsis was present, and after removal of the tonsils there was almost immediate cessation of the dyspepsia and a gain in weight of several stone.

The frequent recurrence of right-sided pain is a familiar story. The association with the erect posture and with physical tiredness, the postural defects, constipation *et cetera*, all make the diagnosis of right-sided ptosis easy. In the later stage of ptosis, but emphatically not in the earlier years, a more or less serious neurosis is frequently associated. The symptoms with a postural relationship can be separated by a careful history from those apparently of nervous origin.

A story of much belching leads one to presume the habit of air swallowing. Such habit formation is most common in nervous dyspeptics, but factors not usually associated with a neurosis, such as pregnancy, are sometimes the apparent ætiological factor. Frequently, I think, the origin of belching is the production of a minor grade of the reverse peristalsis syndrome, and its continuance lies in the fact that belching is a process of air swallowing rather than the reverse.

Vomiting.

Vomiting occupies a prominent place as a dyspeptic symptom. In any acute inflammatory process it is an expression of the reverse peristalsis syndrome elicited by a heightened sensitivity in some lower part of the intestinal tract.

In the more chronic types of dyspepsia it is seen as follows: (i) as an induced state, (ii) in high grade stenosis, (iii) in chronic duodenal ileus, (iv) nervous dyspepsia.

In gastric ulcer, while considerable relief is afforded by acid neutralization and gas production, vomiting usually affords the most complete relief. So much is this so that many patients prefer to induce vomiting in various ways rather than put up with the severe discomfort they would otherwise experience.

As a corollary, the induction of vomiting signifies a very direct stimulus and therefore almost always an organic one, usually ulcer.

A patient with intestinal blockage, from the cardia downwards, will vomit. This blockage may be stenotic from scarring, as in hour-glass stomach or chronic duodenal ulcer; it may result from invasion of the lumen by growth, as in carcinoma at the pylorus; or it may be spasmodic functional blocking, as occurs during the active phases of ulceration, or in chronic duodenal ileus, in which condition an irritable focus seems to exist in the distal duodenum, so that peristalsis stops there and even reverse peristalsis may be initiated.

Vomiting in association with dyspepsia occurs *par excellence* in the nervous dyspeptic. In these sensitized individuals nausea and retching may be produced by the most trivial stimulus, and in a large number this goes on to actual vomiting. In a nervous dyspeptic it is rather characteristic that even if vomiting occurs very soon after meals, there is little interference with nutrition.

Morning vomiting, when the vomit contains much mucus, is characteristic of the earlier stages of gastritis. In this disease of the stomach wall, to which great attention has been paid in recent years, and which is considered by Continental surgeons as an essential concomitant of gastric and duodenal ulcer, over-activity of secreting cells, with excess of mucus formation, occurs in the early phases.

As the gastritis becomes more severe this functional excess is replaced by atrophy, and absence of acid, absence of pepsin and, finally, absence of the essential gastric factor of Castle may occur, the patient ending up with pernicious anæmia.

Excess mucus therefore signifies an early stage in this process and, it may be added, a treatable stage.

Hæmatemesis.

The occurrence of hæmatemesis is often considered the final proof of the presence of a chronic peptic ulcer. This is far from being the case.

Radiologists are well aware that when a patient is referred with a history of hæmatemesis without dyspepsia, no abnormality as a rule is discovered on examination. In other words, unless the dyspeptic history that we have learned to associate with chronic peptic ulcer is present, the occurrence of hæmatemesis does not make the disease any more likely.

The presence of any of the blood states which favour bleeding must first be excluded. Next, evidence of liver cirrhosis should be sought and the patient's consumption of alcohol carefully inquired into. If these two states can be excluded, we can suppose with reasonable certainty that the bleeding comes from either an acute or a chronic ulcer. These two processes are very different and possibly have no relation to each other.

Acute ulceration is definitely related to sepsis and the influence of gastric acidity is not marked.

Chronic ulceration is related to acidity always and the influence of sepsis is uncertain. Bleeding occurs in a very considerable percentage of chronic ulcers, perhaps 10%, and is usually associated with a period of activity of the ulcer, that is, of symptom production; and it is rather typical of the bleeding to produce a remission of the disease if the patient survives.

Much more common, in my opinion, is bleeding from acute ulceration associated with chronic sepsis and, in Brisbane especially, with chronic cholecystitis. I have seen examples also of hæmatemesis related to tonsillitis, to chronic pyelitis, and to residual appendicitis.

Physical Examination.

First, a glance should be made at the general physical type, especially noting whether it seems to correlate with the symptom type suggested by the history.

A complete physical examination should then follow. Nervous excitability should be noted, with the presence or absence of dental or tonsillar sepsis, the state of the pelvic organs, and especially a rectal examination should not be neglected. The state of the thoracic organs should be investigated, and finally the physical overhaul should terminate with a careful examination of the abdomen.

Physical examination of the abdomen is fraught with difficulties. I have no hesitation in saying that nowhere in medicine are more mistakes made than by the erroneous interpretation of signs revealed by palpation of the abdominal wall. I refer especially to the mistaken interpretation of tenderness.

It is interesting to ask oneself what exactly he understands by the term "tenderness". I think you will agree that the term denotes one of two things: it may be either (*a*) pressure pain, the increase of tension produced by pressure increasing an already present pain or bringing a previously latent pain up to the level of consciousness, or (*b*) hyperæsthesia. Here the impulses passing along a nerve of sensation are so heightened by some change in the nerve function that touch becomes interpreted as painful hyperæsthesia, and pressure as severe pain. Note that these conceptions are essentially different in that one denotes a local origin, whereas (*b*) is a purely nervous affection. Outside the abdominal cavity the association of tenderness with inflammation is so constant and common a finding and, moreover, tenderness so accurately localizes the site of such inflammation that there is a universal tendency to consider that tenderness elicited by palpation of the abdominal wall in like manner localizes an underlying lesion. Yet this is not the case. Even if the tenderness can be regarded certainly as being not due to hyperæsthesia, there is very much difficulty in deciding what localizing or other value it possesses.

I should like you to think of the abdomen as a sac lined with a membrane as sensitive to certain stimuli as the cornea of the eye is to touch, every segment of this membrane being joined up by a pathway passing through the spinal cord with a corresponding area of skin. Invaginated into this sac, and thus suspended from one wall by a mesentery, we have certain hollow organs. These hollow organs, in virtue of their protected position, contain no sense organs for the common impulse of heat, touch *et cetera*, and a conscious response can be elicited from them only by sufficient changes of tension in the smooth muscles of their walls. This response constitutes intrinsic pain, and since intrinsic pain is produced by increasing tension, so the only response of the gut to palpation from outside can be an increased production, or tendency to production, of the same intrinsic pain, in the same site as it would occur when produced by causes other than such palpation.

Now for any one portion of the intestinal canal, say for instance, the appendix, where is this site?

It is fundamental here to recall that the intestinal tract is primarily a median tube, slung from the posterior abdominal wall by a dorsal mesentery. In the course of its subsequent lengthening it becomes convoluted, and even in certain parts acquires some mild fixation to the abdominal wall, but as regards its response to stimulation, it retains its primitive pattern and the pain response is felt over the area of the dorsal attachment of its primitive mesentery.

In the case of the appendix, which belongs to the post-arterial half of the mid-gut loop, this response is expressed as a diffuse pain centred on the mid-line, just above and around the umbilicus. Just as all intrinsic intestinal pains are related to the middle line, so all intrinsic pain or tenderness in the tract will be related to the mid-line. The appendix therefore cannot be said to be tender unless palpation over it elicits a pain above or around the umbilicus.

When one palpates the right iliac fossa and the subjective sensation of tenderness is produced, how then must we interpret this sign if it is not an expression of tenderness in the underlying viscus?

I have remarked on the extreme sensitivity of the lining membrane of the abdomen to certain stimuli. This is such that it reacts painfully even to the touch of a bubble of gas, as all who have insufflated Fallopian tubes are aware; for when these patients assume the erect posture a quite severe pain is felt around the tips of the shoulders, which constitutes the skin area related to the diaphragm. It reacts also to certain other stimuli, in particular irritation by intestinal contents (exceedingly), to free blood (less) in contact with it. It reacts to acute or subacute inflammation of a viscus in near contact, the inflammatory state apparently radiating a stimulus to the membrane rather than itself spreading to it, at any rate in the earlier stages. Finally it reacts in a mild degree to excessive distension or to excessive intestinal contraction.

If we exclude that intrinsic intestinal pain which is centred on the mid-line, abdominal pain and tenderness are shown to involve a peritoneo-cutaneous reflex arc,[2] the peripheral limb of which

embraces the identical nervous structures involved in the functional derangement that produces hyperæsthesia.

The local physical stimulus and the general nervous stimulus both end in the same common path.

The physical stimulus may often elicit a more localized response than the neurogenic, but any diagnostic importance this may possess is more than dissipated by the fact that certain particularly tender spots in cutaneous hyperæsthesia closely overlie important intraabdominal organs. These are those points along the outer edge of the rectus sheath where the intercostal nerves are quite bare of muscular covering, and at their most superficial plane.

Since one of these spots is McBurney's point and another the usual site of tenderness in gall-bladder disease, we begin to appreciate that the finding of tenderness is not an unmixed blessing. I can only say that it is a sign that demands the most careful interpretation, and one, moreover, that should never be allowed to dominate impressions gained from other methods of examination.

In making a complete physical examination of the abdomen one will not neglect to note the presence or absence of masses related to the stomach or gall-bladder, while the mobility of the right colon and the ease with which it can be induced into spasmodic contraction should be determined. In these difficult cases no possible evidence deduced from physical examination can be neglected.

Two important methods of diagnosis still remain: (i) the test meal investigation and (ii) radiological examination.

The Test Meal.

Like physical examination by palpation, chemical investigation of the stomach contents will reveal valuable information only if its limitations are recognized.

The principal points to note in a test meal are: (i) the quantity and character of the resting juice, (ii) the emptying rate of the stomach, (iii) the free acid content of the stomach, (iv) the presence of mucus or bile.

The character of the resting juice is probably of the first importance. The presence of charcoal ingested the night before, a foul odour, large quantity of secretion, and perhaps even cell *débris* marks the ulcerating cancer and practically clinches the diagnosis. Old blood helps materially the diagnosis of ulceration, while, conversely, clear fluid in normal amounts is strong evidence against any marked ulcerative or neoplastic change in the stomach.

Emptying time is indicated by the disappearance of starch from the specimens or by the amount of stomach contents at the end of the test compared with the beginning.

The free acid content of the stomach is often of negative rather than direct value. We have already stressed that the acid curve belongs to the type rather than to the disease. There can therefore never be any such thing as a normal curve. The absence of free acid, however, will render the diagnosis of chronic peptic ulcer very unlikely, while, on the other hand, its free presence will render the diagnosis of pernicious anæmia or primary achlorhydric anæmia untenable.

The presence of free acid does not reduce the probability of a carcinoma being present. The acid content, it must again be impressed, depends on the type, and a carcinoma in a hypersthenic individual is compatible with a high acid curve until (i) such extension occurs that the acid secreting surface of the stomach becomes largely involved, or (ii) the associated discharges partly or completely neutralize the free acid.

The presence of a gastric or duodenal ulcer temporarily reduces the free acid content of the stomach because of the associated gastritis present, and its cure is exemplified by a corresponding rise in the free acid curve.

Abundant mucus points to the earlier (and perhaps curative) stages of a gastritis. So we see that the test meal has very distinct values, but rather different values from those sometimes held in the past. My personal experience is that in a doubtful carcinoma and in a mucous gastritis it finds its highest usefulness.

The X Ray Examination.

And finally the X ray examination. Here again, as in all physical methods of examination, the fullest knowledge of abdominal mechanisms is essential, or the most dreadful mistakes will inevitably be made. But granting this knowledge, and in technically expert hands, a modern X ray investigation forms the final court of appeal. The previous investigations may sift the symptoms to a probable diagnosis or brace of diagnoses, but it is finally the radiologist who says that this lesion is present or is not present. Here, finally, the gall-bladders are separated from the duodenal ulcers and the carcinomata from the visceroptoses.

And since the radiologist is all-embracing here too, the patient with phthisis has his or her dyspepsia classified, while (assisted by the electrocardiograph) those supremely difficult problems as between the heart and subdiaphragmatic organs, especially the gall-bladder, are decided.

Treatment.

Compared with the diagnosis, the treatment of chronic dyspepsia is relatively simple. Once the diagnosis is made, treatment is often obvious. As far as the pure gastric symptoms are concerned, certain general principles of treatment are widely applicable.

In particular a smooth diet should usually be prescribed, and the meals should be increased in number and reduced in size. In all conditions where the hypersthenic diathesis is present in association with symptoms it is advisable to reduce the high acid content of the stomach and to counteract rapid emptying by regular two-hourly feeds. The giving

of alkalis is of some value, but is subsidiary in importance to the maintenance of a constant food residue in the stomach.

The treatment of the frequent symptoms associated with visceroptosis and hypotonicity demands a mention. All the secondary factors which tend to support the unfixed caecum should be brought into play. In particular, regular exercises designed to improve posture and to improve tone of the lumbar and abdominal muscles will accomplish wonders. Special attention should be paid to the bowel function, and laxatives of the cascara and paraffin type must be prescribed.

The nervous instability which accompanies the more advanced grades of this condition must be treated by reassurance as to the nature of the complaint, and by bromides.

It is only when all these means fail that any operative intervention should be advised. In our practice not more than 1% or 2% of all visceroptotics are subjected to operation.

The treatment of the nervous dyspeptic must be primarily directed towards the mental factors present.

CONCLUSION.

In conclusion I hope I have interested you in the problems of gastric diagnosis brought forward tonight.

Two things especially I should like to think you will find of permanent value: (i) the idea of diathesis or type, and the light it throws on the type of dyspeptic response, the radiological and the test meal findings; and (ii) the study of the mechanisms underlying abdominal tenderness and the avoidance of false diagnosis that its better understanding permits.

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DIABETES.

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IN two years and five months, from January 1, 1931, to May 31, 1933, when my connexion as medical superintendent with the hospital came to an end, 487 diabetics were treated by me at the Coast Hospital, Sydney. Of these, 417 were admitted to hospital, some several times, and 70 were treated as out-patients only. That so many diabetics should have come to one of the several large metropolitan hospitals is remarkable evidence of the prevalence of the disease. This paper is a record of some details of this large series.

In Tables I and II are shown the age, sex and mortality rate for patients admitted to hospital, and the age of onset of the disease for the whole series.

Females were twice as numerous as males, and 71% of the patients were over forty before any sign of diabetes was observed.

These figures are in harmony with the records of Joslin and of other observers.

For comparison I have extracted the mortality figures published in the Statistical Register of New South Wales.

It should be noted that in the Statistical Register deaths of which tuberculosis or cancer has been a contributory cause are listed under those headings and not under diabetes.

In these death tables the proportion of females to males is not so high as I found it to be in my series, and the age grouping table shows that deaths at ages over forty years were more than 90% of the total. In my series deaths in patients over forty years were 84% of the total deaths. The difference in these percentages may be attributable in part to the exclusion by the Government Statistician of deaths in which tuberculosis or cancer was a factor. The 88 deaths in my series were due to the causes shown in Table V.

Most of these causes of death are not specifically diabetic. Many are complications incidental to advancing age. The essentially diabetic causes in the list are coma, gangrene, carbuncle and hypoglycæmia.

Coma.

Thirty patients were admitted to hospital in coma, and of these 16 died. Table VI shows the age, sex and result of these 30 patients with coma.

Most of the 30 patients in coma were diabetics of twelve months' standing or more, whose coma had been precipitated by some acute cause or by wilful neglect of treatment. But in the following four instances diabetes was not suspected until coma supervened.

CASE XCIX.—The patient was a female, aged forty-five years. On June 27, 1931, a right plantar abscess was incised. On June 29, 1931, the patient became comatose, and on July 1, 1931, at 3 p.m., she was admitted to hospital. On admission she was unconscious and flushed, had air hunger, and exhaled acetone. The urine contained sugar, acetone and diacetic acid, and the blood sugar was 414.

The patient was given an enema and gastric lavage was used. Eight grammes of sugar in a hot drink and 20 units of insulin were administered every half hour till 40 grammes of sugar and 100 units of insulin had been given. During the night 40 grammes of sugar and 100 units of insulin were given.

On July 2, 1931, at 9 a.m., the urine still contained sugar, acetone and diacetic acid. The blood sugar was 241. The patient could now answer questions. In the next twenty-four hours 160 grammes of sugar and "Benger's Food" were given, with 20 units of insulin every two hours (240 units).

On July 3, 1931, at 9 a.m., the blood sugar was 186. In the urine there was no sugar, but a trace of acetone. The patient's condition was much better and she was ordered three pints of milk and 20 units of insulin every four hours.

On July 4, 1931, the urine contained no sugar and no acetone. One hundred units of insulin were taken in twenty-four hours.

On July 5, 1931, the insulin dosage was reduced to 80 units. The patient felt very well.

TABLE I.

Age, Sex and Mortality of Admitted Patients.

Age.	Males.			Females.			Both Sexes.		
	Admitted.	Died.	Mortality *per centum.*	Admitted.	Died.	Mortality *per centum.*	Admitted.	Died.	Mortality *per centum.*
Years.									
Under 20	8	2	25	8	1	12	16	3	19
21 to 40	34	7	21	43	4	9	77	11	14
41 to 60	50	6	12	135	20	15	185	26	14
61 to 80	39	13	33	99	35	35	138	48	35
Over 80	—	—	—	1	—	—	1	—	—
Total	131	28	—	286	60	—	417	88	—

TABLE II.

Age at Onset of all Patients.

Age Period.	Patients Admitted.			Out-Patients.			Total.		
	Male.	Female.	Both.	Male.	Female.	Both.	Male.	Female.	Both.
Years.									
Under 10	5	1	6	1	2	3	6	3	9
11 to 15	1	5	6	—	2	2	1	7	8
16 to 20	4	7	11	2	1	3	6	8	14
21 to 25	6	2	8	1	1	2	7	3	10
26 to 30	13	11	24	—	—	—	13	11	24
31 to 40	15	33	48	2	8	10	17	41	58
41 to 60	56	158	214	17	22	39	73	180	253
61 to 80	27	55	82	2	9	11	29	64	93
Not stated	4	14	18	—	—	—	4	14	18
Total	131	286	417	25	45	70	156	331	487

TABLE III.

Deaths from Diabetes in Ten Years, 1923–1932.

Sex.	1923.	1924.	1925.	1926.	1927.	1928.	1929.	1930.	1931.	1932.	Total.	Average.
Males	131	123	115	94	110	111	129	98	126	141	1,178	118
Females	167	140	155	169	208	174	186	174	263	237	1,873	187

TABLE IV.

Ages at Death, 1931 and 1932.

Group.	Under 5 Years.	5 to 9 Years.	10 to 19 Years.	20 to 29 Years.	30 to 39 Years.	40 to 49 Years.	50 to 59 Years.	60 to 69 Years.	70 to 79 Years.	80 Years.
1931										
Metropolis	—	2	6	3	7	15	32	63	59	6
Remainder of State	2	4	6	3	4	14	41	60	49	13
Total	2	6	12	6	11	29	73	123	108	19
1932										
Metropolis	1	—	4	3	10	15	39	65	58	9
Remainder of State	1	3	9	7	9	16	34	55	32	8
Total	2	3	13	10	19	31	73	120	90	17

On July 30, 1931, she was discharged from hospital feeling very well. She had ceased taking insulin and the blood sugar was 132.

Case CCXLII.—The patient was a female, aged twenty-two years. She was admitted to hospital on May 19, 1932, having been sent in with a diagnosis of "acute abdomen". Diabetes had not been diagnosed, although she had had polyuria and had lost weight in the previous three weeks. When admitted at 2 p.m. she was just comatose and exhaling acetone. The urine contained sugar and acetone, and the blood sugar was 384. There had been acute pain in the region of the liver and up to the right shoulder and neck.

Gastric lavage was performed and sweetened coffee was left in the stomach. Eighty units of insulin were given at once and 120 units during the night.

On May 20, 1932, at 8 a.m., the blood sugar was 70. An ischio-rectal abscess was opened. Probably this abscess had precipitated the coma.

On June 17, 1932, the patient was discharged feeling very well and taking 24 units of insulin daily.

TABLE V.
Causes of Death.

Cause of Death.	Males.	Females.	Total.
Diabetic coma	7	9	16
Diabetic gangrene	—	12	12
Nephritis	5	3	8
Pulmonary tuberculosis	4	3	7
Heart disease	2	11	13
Lung disease, not tuberculous	2	5	7
Cancer	1	3	4
Pancreatitis	1	1	2
Senility	1	2	3
Epilepsy	—	1	1
Cerebral syphilis	1	—	1
Cerebral abscess	1	—	1
Abscess	—	3	3
Carbuncle	1	3	4
Liver cirrhosis	—	1	1
Cerebral hæmorrhage	—	2	2
Meningitis	—	2	2
Hypoglycæmia	—	1	1
Total	26	62	88

TABLE VI.
Patients in Coma.

Age.	Males.		Females.		Both Sexes.	
	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.
Years.						
Under 10	—	1	—	—	—	1
11 to 15	—	—	—	—	—	—
16 to 20	—	1	2	—	2	1
21 to 25	—	—	2	—	2	—
26 to 30	1	—	1	—	2	—
31 to 40	—	3	1	1	1	4
41 to 50	1	—	3	3	4	3
51 to 60	—	1	3	3	3	4
61 to 70	—	—	—	1	—	1
71 to 80	—	1	—	1	—	2
	2	7	12	9	14	16[1]

[1] Of these fatal cases, nine occurred within twenty-four hours of admission to hospital.

CASE CCLXIII.—The patient was a male, aged thirty-four years. He was admitted to hospital on June 17, 1932. On the previous day, at another hospital, a tooth had been extracted and a pile ligatured under ether anæsthesia. That night, at his home, he had pain in the chest and breathlessness. He was sent to the Coast Hospital with a diagnosis of "pulmonary infarct". Diabetes had not been diagnosed, though there was a history of polyuria for some months. On admission he had marked air hunger with sugar and acetone in the urine. In spite of active treatment the weak pulse gradually failed and the patient died eleven hours after admission.

CASE CCCXLIII.—The patient was a male, aged twenty-eight years. He was admitted to hospital on Christmas Day, 1932, at 11.30 a.m. He had had thirst, polyuria and loss of weight for four weeks, and vomiting and air hunger for twenty-four hours. His condition had been diagnosed as diabetes shortly before admission.

On admission to hospital the patient was cyanosed, with marked air hunger. He was exhaling acetone and vomiting frequently. The blood sugar was 543 and the urine contained much sugar, acetone and diacetic acid.

He was at once given 32 grammes of glucose (most of which he promptly vomited) and 40 units of insulin. Forty units of insulin were again given at 3 p.m. and at 7 p.m., and the patient took abundant sweetened drinks, much of which he vomited. At 11 p.m. and 2 a.m. the urine was sugar free and scanty (only 30 cubic centimetres or one ounce was passed during the night).

On December 26, 1932, at 8 a.m., the blood sugar was 280, the urine was sugar-free, and the patient was still vomiting. No more insulin was given.

On December 27, 1932, his condition had improved, but he was still vomiting occasionally. In twenty-four hours 360 cubic centimetres (twelve ounces) of urine were passed; there was a trace of glycosuria.

After this the patient steadily recovered and was discharged from hospital on January 25, 1933.

Subsequently, in May, 1933, he had an hæmoptysis, and X ray examination revealed a "tubercular lesion of the right upper lobe".

In another instance tooth extraction was the exciting cause of coma.

CASE CCCLVII.—The patient was a female, aged sixteen years. She was admitted to hospital on February 1, 1933, at 11 p.m. in semi-coma with marked acidosis. She had had diabetes for two years and was being treated with insulin as an out-patient of another hospital. That morning she had had a tooth extracted and coma began at about 4 p.m.

On admission to hospital the blood sugar was 458 and the urine contained sugar and acetone. The patient was exhaling acetone and breathing deeply, but without actual air hunger. Glucose and insulin were given freely and her condition improved rapidly. Thirty-three hours after admission the morning blood sugar was 124. The insulin dose was then reduced to 40 units and she was put on a high carbohydrate diet of 1,200 calories with a fatty acid to glucose ratio of 0·5.

On February 15, 1933, she was discharged feeling well.

It will be noted that all these patients were treated with glucose and insulin. The rationale of this treatment is expounded by Himsworth in *The Lancet* of July 23, 1932. He maintains that "the fundamental idea is to give glucose, glucose always, and covered by the necessary amount of insulin—not insulin covered by glucose". That ketosis and "protein breakdown" diminish or increase as the blood sugar rises or falls. That a "diabetic passes into coma, not because his blood sugar rises, but because it does not continue to rise". He states that at University College Hospital, London, in two and a half years, 41 cases of varying degrees of diabetic intoxication have been treated on these principles, and that the same "steady automatic recovery" has occurred in each case. For the period August, 1932, to May, 1933, I saw 14 cases of diabetic intoxication and treated them all on these principles. Nine made "steady automatic recoveries" and five died. But in these fatal cases the patients were mostly beyond treatment when admitted to hospital, as indicated by the following brief details:

CASE XXIX.—A male, aged seventy-six, died six hours after readmission.

CASE CLXXXIII.—A female, aged seventy-one, with right basal pneumonia, died on the fifth day after admission.

CASE CCXX.—A female, aged thirty-seven, died two hours after admission.

CASE CCCV.—A male, aged thirty-three, died six hours after admission.

CASE CCCLXXXIV.—A female, aged fifty-four years, died twenty hours after admission.

Gangrene.

Thirty-four patients had gangrene, and of these twelve died. All but one (a female) were over fifty years of age, namely: 51 to 60 years, three males, five females; 61 to 70 years, one male, seventeen

females; 71 to 80 years, two males, five females. This age distribution was to be expected, since vascular changes and senility progress together. But I am at a loss to explain the great preponderance of females—28 females to 6 males. Of the admitted males, 4·5% had gangrene; of the admitted females, nearly 10%. This is the opposite of Joslin's experience, according to which gangrene affected twice as many males as females.

Histologically the arteries of amputated legs showed that narrowing of the lumen was due to proliferation of the intima. In some cases the popliteal artery was almost entirely occluded in this way. What causes the endarteritis has yet to be discovered. In my series one might surmise that the females were more liable to this because their less active mode of living made their lipæmia higher than that of the males. I regret that I have no information as to whether they actually did have higher lipæmia than the males and than the other females who did not get gangrene.

Of the twelve fatal cases, six patients declined amputation. Three others were in a hopeless state on admission, namely:

Case XCV.—A female, aged sixty-seven, three days before admission cut a corn. On admission she was comatose, with a gangrenous foot, and died six hours afterwards.

Case CXXII.—A female, aged sixty-five, was admitted in a state of coma with two large areas of superficial gangrene of the abdominal wall. She died twenty hours after admission.

Case CCLXIX.—A female, aged sixty-nine, had a blister below the left knee. On admission there was discoloration up to the gluteal region, with patches of gangrene. She died twenty-four hours after admission.

Thirteen patients had amputation above the knee. Of these, three died in the following circumstances:

Case LXX.—A female, aged sixty-nine, was admitted on April 14, 1931, with gangrene of the right foot, which had followed the cutting of a corn a month previously. Cellulitis and suppuration spread in the foot despite free incisions. There was scantiness of urine and not much glycosuria. After much persuasion the patient consented to amputation above the knee, which was done on May 4, 1931. But her general condition became worse and she died four days later.

Case CCXXXIII.—A female, aged sixty-one, was admitted on April 14, 1932. At another hospital five weeks previously she had had an amputation below the right knee. On admission to the Coast Hospital the stump had not healed, there was much pain, and the fibula protruded from the wound. Next day (April 15, 1932) re-amputation above the knee was performed. This wound healed almost completely, then broke down and discharged fishy smelling pus. The patient died on June 28, 1932. In many estimations of her blood sugar it was never over 159, and generally was about 90.

Case CCXLI.—A female, aged fifty-six, was admitted on May 30, 1932, with oliguria, albuminuria and dropsy, and a history of diabetes for eleven years. A glucose tolerance test confirmed the diagnosis of diabetes. While in hospital she developed gangrene of the left heel which, four days later, had spread to the dorsum of the foot. On October 21, 1932, under spinal anæsthesia, amputation was performed above the knee. Five days later the stump was gangrenous. The patient became comatose and died on October 29, 1932. Sections of the popliteal artery showed almost complete occlusion by *endarteritis obliterans*.

Altogether sixteen amputations were done—thirteen above the knee and three below the knee, of which one was a Chopart amputation and one a Lisfranc. The anæsthetic used was ethylene and oxygen in ten instances, nitrous oxide and oxygen in three, spinal "Neocaine" in two, and open ether in one. The Senior Resident Medical Officer (Dr. C. J. Walters, F.R.A.C.S.) was the operator in every case. He found ethylene and oxygen more satisfactory than nitrous oxide and oxygen—perhaps because we had not a good apparatus for the latter. Spinal "Neocaine" he considered to be best. In no case was a tourniquet used, and there was never any difficulty in controlling hæmorrhage.

Carbuncle.

Twelve patients had carbuncle, and of these four died. This is rather worse than the one in four mortality which Joslin says he expects nowadays. But the following details show that these four patients who died were very unpromising cases.

Case LXXI.—A female, aged fifty-two years, was admitted on April 18, 1931, and died on April 20, 1931. She had extensive carbuncle of the nape of the neck for the previous two weeks; also carbuncle of the left knee and marked acidosis.

Case CXII.—A female, aged seventy years, had a large carbuncle of the left thigh of four weeks' duration. She developed pyæmia, with left cervical abscess, left perinephric abscess and left basal pneumonia.

Case CCCXII.—A female, aged forty-seven years, had a carbuncle of the nape of the neck and acidosis. She died six days after admission.

Case CCCLXVII.—A male, aged sixty-seven years, a frail old man, had a carbuncle of the neck of three weeks' duration. He had no diabetic treatment before admission. There was acidosis and the blood sugar was 508. He died four days after admission.

The nape of the neck was the site of the carbuncle in five patients. This is the commonest site—perhaps from neglect of cleanliness. Joslin quotes: "The washed neck never boils", to emphasize the importance of this. Four other patients had the carbuncle on the lower limb, two on the trunk and one on the upper limb.

In treatment I did not use the free crucial incisions advocated by some, but incised merely to relieve tension or evacuate pus. A dressing of magnesium sulphate and glycerine was found beneficial, the formula being 24 ounces of exsiccated magnesium sulphate to 11 ounces of glycerine. This is worked into a paste and applied on lint. Whenever practicable, Bier's suction glass was used. Some patients were given injections of sterilized milk or of "Aolan" and apparently with benefit.

Hypoglycæmia.

Several patients were admitted in hypoglycæmic coma. One of these (Case CCCXXXVII) died fifteen minutes after admission.

She was a woman aged fifty-three years, who had been discharged three weeks previously from another hospital, where she had been treated for diabetes. Her daughter said she had been taking insulin, but did not know the quantity. Urine obtained by catheter after death contained some acetone, but no sugar.

Other hypoglycæmic patients of interest were:

CASE XCIII.—The patient was a female, aged thirty-three. She had been receiving diabetic treatment for four years, keeping very well, and leading a very active life on 36 units of insulin daily. On New Year's Day she got up at 6 a.m., gave herself 20 units of insulin without having any food, prepared breakfast and did housework. She had done all this without any feeling of illness, but at 8 a.m. suddenly lost consciousness and fell like a stone, bruising the right side of the face. Her husband gave her some sugar and she vomited. When admitted to hospital at about 9 a.m. she was almost conscious, vomiting frequently. The blood sugar was then 88 and the urine was sugar-free. On being given orange juice and sugar the patient quickly recovered and was discharged quite well next day.

CASE CCCXXX.—The patient was a male, aged forty-seven years, who had been under diabetic treatment as an in-patient and out-patient for seven years. On January 9, 1933, just after midnight, he was re-admitted to hospital in deep hypoglycæmic coma. The blood sugar findings were: at 12.15 a.m., 26 (0·026%); at 12.45 a.m. (becoming conscious), 44 (0·044%); at 7 a.m., 310 (0·31%). Being treated with honey by mouth, the patient recovered rapidly and was discharged next day.

In this patient the degree of hypoglycæmia was remarkable, and also the recovery of consciousness when the blood sugar rose to 44. In another instance (Case XLI) the blood sugar was 40 during unconsciousness and 60 as the patient became conscious.

CASE CCCLXVII.—The patient, a female, aged twenty-eight years, had been under diabetic treatment as an in-patient and an out-patient for eight years. She was readmitted to hospital on March 27, 1933, in profound hypoglycæmia. She had taken 35 units of insulin before breakfast and had felt "queer" at 9 a.m. Then she ate an orange and lay down. At 2.30 p.m. her husband came home and found her in deep coma. On admission to hospital at 3.30 p.m. she was still comatose and the urine contained acetone, but no sugar. Under treatment with honey she recovered in two hours and was discharged two days later.

This same patient had been last admitted to hospital three weeks previously, on March 4, 1933. She was seven and a half months pregnant and under treatment by her own doctor was taking 70 units of insulin and a low carbohydrate diet. She came to hospital on account of hypoglycæmia, and I suspended insulin for twenty-four hours. The result was a blood sugar reading of 384, air hunger and abdominal pain. With glucose and insulin this patient made a rapid recovery.

This case illustrates the tendency to hypoglycæmia in pregnancy, which has been observed by others, and also the rapidity with which such a patient may develop acidosis if insulin be abruptly omitted. Joslin emphasizes the value in pregnancy of high carbohydrate diet and of comparatively small and frequent doses of insulin.

Besides these patients, many others had hypoglycæmic reactions, and there were instances of all the symptoms described in the text books. Diplopia was common, with sweating and a "nervous feeling".

In Case CCCXXX the patient, in one reaction, clenched his fists spasmodically and was very restless. In Case LIA the patient behaved very strangely, wandering about the ward and peering under the beds. In Case CLXXIX the patient, a boy, aged eleven years, was several times found unconscious in the hospital grounds. These attacks ceased when he was put on a higher carbohydrate diet. In Case CLXXXI the patient, another boy aged eleven years, was for several hours unconscious, violently restless and making grunting noises. Honey and the hypodermic injection of adrenalin brought him round.

PROSTATISM.[1]

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I HAVE been asked to make some observations tonight on the indications for operation in patients with prostatic disease; to endeavour to make clear at what stage of the disease operation should be performed; and to say a few words on the preparation of these patients for operation. Unfortunately, most of us in our early professional life witnessed the results of prostatectomy in the wards of our big metropolitan hospitals, and inevitably we were forced to the conclusion that prostatectomy was an operation of absolute necessity, to be postponed as long as possible, and strongly to be advised against until every other expedient had been employed and had failed.

To combat this unfortunate though perhaps natural reaction there are two facts I should like you to consider at the outset. Firstly, prostatic disease is invariably a progressive condition, whether it be malignant or non-malignant. Hence an early operation will save untold suffering later. Secondly, the mortality rate following on prostatectomy performed by competent urologists should not exceed 5% to 6%, and is, in fact, in our department at the Lewisham Hospital nearer 3%. This low mortality rate, I think you will agree, is an almost irreducible minimum for any major surgical procedure.

It is now the generally considered opinion of urologists that once surgical prostatic disease has become manifest, prostatectomy cannot be performed too early. To elaborate this statement, I shall briefly outline a typical prostatic history and endeavour to indicate the dangers of delaying operation.

Of the early symptoms, the most commonly complained of are nocturnal frequency and diminution of the force of the urinary flow. Dribbling at the end of micturition and delay in commencing the act are also common. Hæmaturia is rarely an early symptom. Repeated gross bleeding in prostatic patients may be due to an intravesical growth, and these patients should be examined by the cystoscope, even in the presence of a palpably enlarged prostate. Renal pain, which has been well named "reflux renal colic", is sometimes an early though by no means a common symptom. It must be remembered, however, that all these symptoms may occur intermittently.

[1] Read at a staff meeting held at the Hornsby and District Hospital, November, 1933.

It is opportune here to point out that there are many cases of prostatic enlargement that cannot be diagnosed by rectal examination. These are readily demonstrable, however, by cystoscopic inspection, which should be carried out in all doubtful cases. This applies particularly to that group known as posterior commissural obstruction, including true middle lobe enlargement, hypertrophied Albarran's glands, and inflammatory median bar formation, the so-called fibrotic prostate. I need scarcely recall to you those cases of sciatica due to nerve involvement in malignant prostatic disease in order to emphasize the importance of a routine rectal examination in all patients.

With regard to age, the earliest symptoms are relatively common after the age of fifty years, but it must be remembered that some considerable degree of prostatic hypertrophy may occur in men under fifty years of age.

As the growth of the prostate proceeds, increasing frequency and difficulty in micturition occur; complete evacuation of the bladder becomes impossible and attacks of acute cystitis and pyelonephritis may occur. A vicious circle is set up. Calculi may form in the infected residual urine.

I should like to sound a note of warning here. Vesical calculi in patients of prostatic age are almost always the result of bladder neck obstruction, and it is generally unwise to perform an operation for their removal, either lithotrity or cystotomy, disregarding the primary cause of their formation, namely, the enlarged prostate gland. Of course, as an exception, there are those calculi that have definitely descended from the upper urinary tract. These may require operative removal.

Once residual urine has developed, further postponement of operation is likely to be followed rapidly by increasingly frequent and difficult micturition, intermittent attacks of retention of urine and of pyelonephritis. Ultimately, complete retention of urine accompanied by renal and cardiac insufficiency will urgently demand surgical relief. Unfortunately, before this can be accomplished with safety, a long period of pre-operative hospital treatment is only too often necessary.

From the foregoing remarks it will be gathered that the presence of residual urine is an outstanding indication for operation. By residual urine, it will be understood that a residuum can be withdrawn by catheterization, after a voluntary attempt to complete bladder evacuation has been made by the patient. In the Urological Department at the Lewisham Hospital it has long been the standard practice that when four ounces or more of residual urine are present, pre-operative preparation by an indwelling catheter must be carried out.

Inguinal herniæ and hæmorrhoids are commonly found in prostatic patients. It is generally inopportune to attempt to rectify either of these conditions while any degree of urinary obstruction is present.

Some words now on the pre-operative preparation. I shall introduce this, with a rather full description of urethral catheterization, partly on account of the extreme importance of employing an aseptic technique and partly owing to the all too frequent neglect of reasonable aseptic and antiseptic precautions. When it is realized that the so-called urinary chill following urethral instrumentation is in reality a bacillæmia, and that the prostatic urethra has the unique power of absorption of organisms directly into the blood stream, I think we are more than justified in taking extreme precautions in such manipulations. In addition, failure in asepsis during this stage of preliminary catheter drainage is a potent cause of post-operative sepsis.

Before proceeding to catheterization in hospital every prostatic patient is shaved in the suprapubic and scrotal regions. Immediately before the catheter is passed the patient empties his bladder as completely as possible. The penis is cleaned with methylated spirit. The urethra is then freely syringed out with a solution of 1/10,000 oxycyanide of mercury, by means of a glass syringe. The catheter, freely lubricated with an antiseptic lubricant, is then passed with forceps without at any time coming in contact with the operator's hands. When less than four ounces of residual urine are present the renal function test is done—eight cubic centimetres of a 0·4% solution of indigo-carmine are injected intravenously. If the result of this test is satisfactory, and if the patient's general condition warrants it, operation is undertaken after two or three days' rest in hospital without any preliminary catheter drainage. When a residual urine of more than four ounces is found, when the indigo-carmine test is unsatisfactory, or when the urine shows gross infection, an indwelling catheter is employed for at least ten days. A retained catheter is always to be preferred to frequent and repeated single catheterizations, owing to the diminished risk of infection.

When the general condition of the patient is unsatisfactory, even in the presence of a normal renal function test, a remarkable improvement is frequently effected by employing an inlying catheter.

In all cases that have not been submitted to recent instrumentation a bilateral ligation of the *vasa deferentia* is performed as a routine measure. This has greatly lowered the previous incidence of epididymitis, both as a pre-operative and as a post-operative complication.

More than six ounces of urine should not be withdrawn at any one time from a distended bladder. Gradual decompression should be the rule. After this amount has been removed the catheter is clamped. A short length of rubber tubing, closed at the free end, is attached to the catheter, and an ordinary hypodermic needle (also with rubber tubing attached) is made to perforate into the lumen of the catheter tubing. The urinary outflow through this needle is adjusted so as to drain at the rate of four ounces per hour into a sterile bottle.

During this period of decompression particularly, and during the whole of the pre-operative and the greater part of the post-operative period, fluids by

ILLUSTRATIONS TO THE ARTICLE BY DR. N. D. ROYLE.

FIGURE IV.

FIGURE V.

FIGURE VI.

FIGURE VII.

FIGURE VIII.

FIGURE IX.

FIGURE X.

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mouth must be pushed to the limit of tolerance, due consideration being given to the maintenance of a balance between fluid intake and cardiac efficiency.

After the bladder has been completely emptied, and this may take as long as 24 to 36 hours, the needle is removed and the tubing is attached to the catheter and is allowed to drain freely into a bottle fixed to the side of the bed. Care must be taken that the end of the connecting tube is always kept below the level of the antiseptic fluid in the bottle, one in twenty carbolic lotion, to insure a continuous siphon action and thus uninterrupted drainage. The open end of the catheter is never in any circumstances allowed to lie in a urinal. When this has occurred infection has invariably resulted, and it has been found absolutely impossible to clean up a dirty bladder under these conditions. The catheter is replaced every three days and, if the patient is well enough, he is given a hot bath whilst the catheter is out.

Bladder irrigation is performed, depending upon the grade of infection of the urine, once, twice or even three times a day. A pale pink solution of permanganate of potash is repeatedly syringed into the bladder; this is completely removed by washing out with sterile water, and finally 120 cubic centimetres (four ounces) of a 1/3,000 solution of silver nitrate is run into the bladder and allowed to remain for half an hour. If the urine should be very grossly infected, the strength of the silver nitrate is increased even up to 1/1,250, but should not be so strong as to cause the patient any discomfort. By these means, with extremely few exceptions, it has been found possible to clean up even the most filthy bladder.

A mixture containing ammonium benzoate (0·9 gramme or 15 grains) is given four times a day, and hexamine tablets (0·6 gramme or 10 grains) three times a day. This medication is a routine measure throughout the hospital period, excepting that hexamine is withheld whilst any blood is present in the urine.

When the patient has become accustomed to the inlying catheter—generally after three or four days—he is allowed out of bed in a chair, the catheter tubing still draining into the bottle, and he is, if possible, taken out of doors. This is an important practical point and often hastens the pre-operative recovery. The catheter drainage is carried on for never less than ten days, the duration depending, *inter alia*, on the results of the indigo-carmine tests, which are repeated at weekly intervals. From the time of the patient's admission until his discharge from hospital a physician is in consultation.

As an alternative to an indwelling catheter, suprapubic cystotomy may be performed. As a routine procedure we do not favour two-stage prostatectomy. There are, however, certain definite indications for its adoption. They are: (i) Failure to tolerate an indwelling catheter. This, in our experience, is rare. (ii) Repeated attacks of pyelonephritis or intractable epididymitis during the preliminary stage of catheter drainage. (iii) Delayed recovery of renal function after a reasonable period of catheter drainage (three to four weeks). And finally, (iv) when the patient's general condition is such that prostatectomy is for the time being contraindicated.

Prostatectomy is never undertaken after a preliminary cystotomy until a period of at least four weeks has elapsed, as this allows induration around the wound to subside.

In conclusion, may I plead for the early recognition of the prostatic syndrome; for the importance of recommending early operation, especially in the presence of residual urine; and for the education of the public to combat the popular fallacy that these distressing symptoms are the normal accompaniments of advancing years?

THE TREATMENT OF UNUNITED FRACTURES.

By N. D. Royle, M.D., Ch.M. (Sydney), F.R.A.C.S.,
Honorary Orthopædic Surgeon, Lewisham Hospital, Sydney; Honorary Demonstrator in Anatomy, University of Sydney.

The problems in treatment of ununited fractures are: (i) to provide a medium by which the fragments may be united, and (ii) to confer rigidity upon the medium and the uniting fragments. These problems are met by the use of massive autogenous bone graft fixed in place by beef bone screws. The beef bone screws are fixed in place by the employment of drill and taps. In the case of the humerus and femur one-quarter inch screws are of sufficient strength; in the forearm three-sixteenth screws are used. It will be seen from a study of the radiographs that the fate of the screws differs little from that of the autogenous graft. The screws are often absorbed and are replaced by new bone, while the grafts actually grow on to the recipient bone. The screws remain fixed long enough, however, for the graft to grow into place.

The Technique.

In the humerus, in addition to conferring rigidity on the endoskeleton, it is necessary to provide an exoskeleton in the form of a jacket of celluloid or some other durable material. Plaster, excepting "Cellona", is not altogether satisfactory for this purpose, though it may be used in the absence of lighter material. The advantage of celluloid is that it may be made into a better finished and more comfortable article. It is made on a casting and so a good fit is assured. My instrument maker, Mr. F. E. Bennett, makes a very satisfactory appliance of celluloid lined with fine felt which is both comfortable and light. (The splint is illustrated in Figure I.)

The Preparation of the Graft and Its Bed.

The massive graft is obtained from the flat anterior surface of the tibia by drilling a number of holes as illustrated (see Figure II) and by

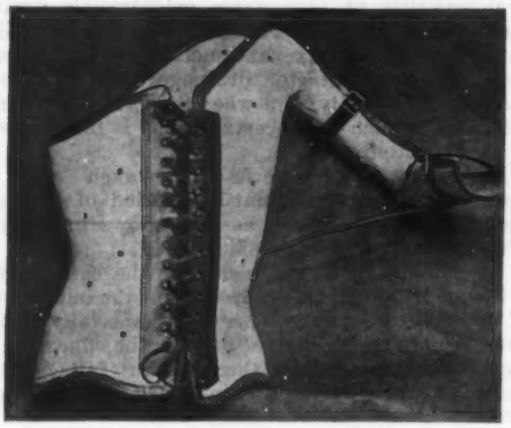

FIGURE I.
Jacket worn after operation for ununited fracture of humerus.

cutting with an osteotome from one hole to the next. This provides a better graft than one cut with an electrically driven circular saw, which sears the edges of the graft. There is also less machinery

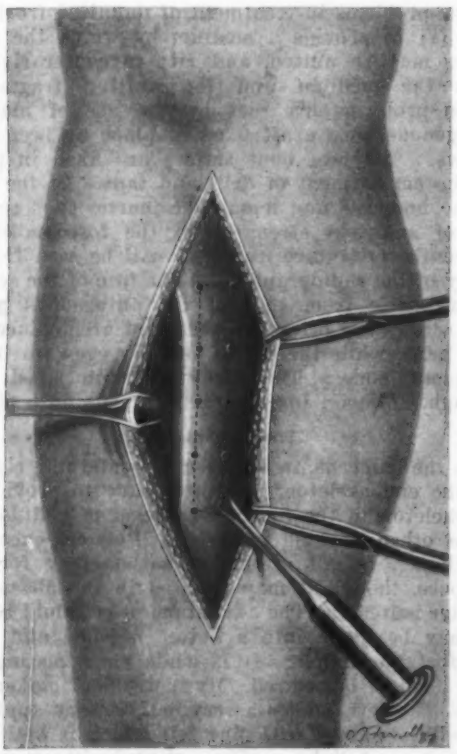

FIGURE II.
Showing method of obtaining bone graft.

in the theatre to be watched. A simple mechanical drill with a universal chuck and standard drills obtainable at any tool store are quite satisfactory for drilling for massive grafts.

For the humerus the graft should be 1·5 to 2·0 centimetres wide, 10·0 centimetres long, and the full thickness of the cortex of the tibia in depth. When both or either of the bones of the forearm require grafting the width of the graft should be 1·5 centimetres and the length at least 8·0 centimetres. The graft for the femur should be 2·0 centimetres wide and 12·0 centimetres long to be effective. It is an advantage, though not essential, to preserve the periosteum intact.

The preparation of the bed must next be considered. There is no need to sacrifice the ends of the ununited fragments. These may be approxi-

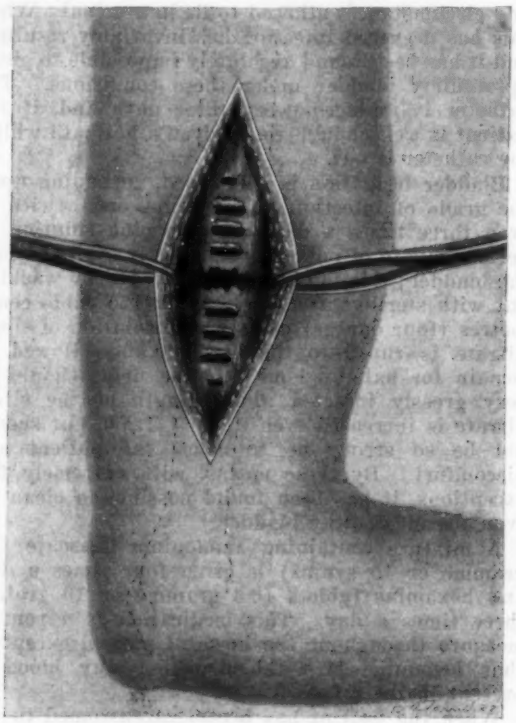

FIGURE III.
Showing rucking of recipient bone of humerus before graft is applied.

mated and freed from scar tissue, but not cut off so as to shorten the limb. With a bone file or an ordinary wood rasp a flat surface is prepared on the most convenient aspect of the recipient bones and the surface rucked up with an osteotome, as follows (see Figure III).

The Application of the Graft and the Conclusion of the Operation.

The graft, held in a small vice, is drilled. The vice may be held by an assistant or screwed on to the table. The size of the clearance holes in the graft is the same as that of the screw to be used. The recipient bone is then drilled with a drill somewhat smaller than the size of the screw. The tap cuts into bone more easily than into metal and a good thread is assured if the appropriate drills

are used. A good plan is to fix one screw first and drill and tap the remainder through the clearance holes of the graft. At least four screws should be used. Care should be taken not to splinter the heads of the bone screws with the screw driver, as these hold the graft in position in close contact with the recipient bone.

In contrast to the inlay graft, the lay-on graft, with fixation by bone screws, makes so firm a job that the limb may be moved with impunity when it is being dressed and splinted.

In the forearm two operations are necessary. One bone only should be grafted at each operation and the ulna should be grafted first. A plaster splint is quite satisfactory for the forearm and should extend from the middle of the arm to the centre of the palm and include the base of the thumb. In fractures of the tibia fixation in plaster of Paris is an easy problem, and the use of the massive sliding graft is to be recommended. There is no need to take the graft from the opposite tibia. The sliding graft may with advantage be fixed in position with bone screws; but good results may be obtained without internal fixation on account of the extremely satisfactory external fixation of the unpadded plaster splint. After a massive graft has been removed from the tibia the leg should be encased in plaster for six weeks to obviate the occurrence of fracture.

Description of Patients' Lesions.

Ununited Fracture of Humerus.

The patient had sustained a gunshot wound of the left arm in May, 1917. The humerus in the distal third was fractured and the radial nerve divided (Figure IV). The injury resulted in an ununited fracture for which the patient received various forms of treatment. The humerus was plated in 1918; a sliding graft was made in February, 1919; an inlay graft from the tibia was applied in October, 1919. When he came under my care the patient already had had nine operations for the relief of his condition. His elbow was ankylosed at about 50° and the muscles supplied by the radial nerve were paralysed. I applied a massive graft from the tibia to the humerus. One month later the radiographic report was as follows: "Commencing union between the graft and the fragments of humerus and also between the screws and humerus and between the two main fragments of the humerus" (Figure V). And seven months later it read: "Bone graft appears to be in excellent position and there is well marked bony tissue between the graft and bone and also well marked bony callus between ends of the fragments." The splint was then discarded. The patient had a subsequent operation for paralysis of the dorsiflexors of the wrist and extensors of the fingers which restored the use of his hand.

Ununited Fracture of Radius and Ulna.

The patient sustained a fracture of the left forearm on July 25, 1929, which resulted in an ununited fracture of both radius and ulna. The patient was first seen on October 27, 1931, when an X ray picture showed both fragments of each bone in close apposition, but ununited. A massive bone graft to the ulna (Figure VI) was applied on November 11, 1931, and to the radius (Figures VII and VIII) on April 15, 1932. The result is shown in the radiograph taken on November 28, 1932 (Figures IX and X).

Reports of Cases.

TORSION OF AN OVARIAN CYST IN A CHILD OF FIVE.

By R. M. Glasson, M.B., Ch.M. (Sydney),
Wellington, New South Wales.

L.P., aged five years, had retired to bed at 6 p.m. on September 4, 1933, and had slept well till awakened at 2 a.m. by pain in the region of the right groin and back. She had vomited then and had had no more sleep, owing to the pain, which at times became worse. Her mother stated that she was convalescent from measles, having been allowed up for the first time on September 4, and that her bowels had acted naturally before she went to bed.

Seen at 9 a.m. on September 5, she was an intelligent child, complaining of pain in the right groin. The temperature was normal and the pulse rate 70, and she did not look ill. Her tongue was clean. The abdomen was normal, there was no tenderness in the right iliac region. The urine was acid and clear. The chest was normal. The pain persisted all day, at times coming on in acute attacks. No further vomiting occurred and the temperature and pulse remained the same.

On the following morning she was admitted to hospital. The abdomen was more distended, but there was still no tenderness or rigidity. The pulse rate was then 96 and the temperature normal. Later in the day she became definitely tender in the right quadrant and the temperature had risen to 37·8° C. (100° F.). At this time a diagnosis of acute appendicitis was made and the abdomen was opened by a McBurney incision. The appendix was normal, but the right side of the pelvis was occupied by a large cyst about the size of a tennis ball. It was delivered with difficulty and removed. Convalescence was uneventful.

Dr. S. R. Dawes reported as follows:

> A number of sections were cut, but nothing to identify the nature of the cyst was found.
>
> The cyst measured approximately five centimetres in diameter and was roughly spherical. It was of a deep black colour, apparently being most congested.
>
> On section the wall of the cyst appeared black. It was up to 0·7 centimetre in thickness, except on one side, where it was 1·5 centimetres. The contents consisted of blood-stained fluid.
>
> Sections show a very loose tissue packed everywhere with blood cells, which have permeated and infiltrated every space, causing the looseness of the organized tissues.

Reviews.

HISTORY OF SCOTTISH MEDICINE.

Once again the medical profession owes a debt of gratitude to the Wellcome Medico-Historical Museum, for with its assistance Dr. Comrie has been enabled to give us the two handsome volumes on the "History of Scottish Medicine".[1] In these two volumes we have set before us a most fascinating study of the history of our profession

[1] "History of Scottish Medicine", by John D. Comrie, M.A., B.Sc., M.D., F.R.C.P.; Volumes I and II (published for The Wellcome Historical Medical Museum); Second Edition; 1932. London: Baillière, Tindall and Cox. Crown 4to., pp. 852, with two plates and 404 figures in the text. Price: 50s. net.

in Scotland. Dr. Comrie takes us back to the primitive ages when surgical instruments consisted of flakes of flint and the operation of trephining was the chief surgical procedure, and passing in review the gradual growth of the famous medical schools and universities, brings his story down to the present day.

In early Christian and Saxon times many quaint and interesting stories are told of the medical skill of Saint Columba (521-597) most of whose cures border on the miraculous. Thus, on one occasion, Saint Columba picked from the river Ness a white stone, by which he said God would effect the cure of many diseases. This stone he sent by two of his disciples to Brude, the pagan king of the Picts, who had requested him to cure his foster-father, Briochan the Druid, then lying in a dying state. The stone was immersed in water, when, contrary to the laws of nature, it floated on the water like a nut or apple and could not be submerged. Briochan drank the water in which the stone was floating and immediately recovered perfect health. The stone, most probably a piece of ambergris, was afterwards preserved among the treasures of the king, retained its miraculous property of floating in water and, as the chronicler says, "through the mercy of God, effected the cure of sundry diseases".

One hundred years later we have Saint Cuthbert healing the sick partly by the knowledge he gained from the medical manuscripts preserved in the monasteries, and partly by miraculous means. Even after the death of the saint his reputation was so great that as the ancient records state, "the miracles which he worked when alive did not cease".

Dr. Comrie gives a most interesting account of monastic medicine and, as an example of a Scot who journeyed to the Continent and attained a reputation as a scholar and a doctor in foreign parts, he gives a long account of Michael Scot, who lived at the end of the twelfth century. So great was his fame as a seer of the future and magician that Dante has immortalized him as:

> That other, round the loins
> So slender of his shape, was Michael Scot
> Practised in every slight of magic wile.

Scot as a practising physician enjoyed a great reputation, being especially celebrated for his treatment of leprosy, gout and dropsy. One of his pills, known as *Pilulae Magistri Michaelis Scoti*, is noted by a thirteenth century copyist as effective to "relieve headache, purge the humours wonderfully; produce joyfulness, brighten the intellect, improve the vision, sharpen hearing, preserve youth and retard baldness". Those modern practitioners who are interested, will find the formula given by Dr. Comrie in his book.

The narrative is enlivened by many references to historical personages, and we read how Edward the First was treated and cured at Torpichen Priory in 1298 after being injured by a kick from his horse. Later on in 1306 the king suffered from an intractable dysentery, most probably due to a malignant growth, and Master Nicholas de Tyngewyk was sent for to treat the king but the patient died in July, 1307 and the doctor's bill amounted to £134 16s. 4d., equivalent to well over three thousand pounds sterling nowadays, for the physician's care, remedies and travelling expenses.

In 1450 the inhabitants of Edinburgh were forbidden to allow swine to wander freely in the streets and this was one of the earliest public health measures, and later in the fifteenth century this was followed by further regulations for the prevention of the spread of infectious diseases.

A very quaint document, of which a facsimile page is given, was "The Seal of Cause" which was granted to the Guild of Surgeons and Barbers and confirmed by King James the Fourth in 1506. Amongst the privileges granted to members of the crafts was the sole right to manufacture and sell *aqua vitæ* within the burgh, and had this monopoly to make and sell whisky in Edinburgh not been allowed to lapse, the Royal College of Surgeons would today have been one of the wealthiest corporations in that country.

The interesting origins and histories of the various colleges of physicians and surgeons, and of the universities and medical schools in Edinburgh, Glasgow, Aberdeen and Saint Andrews are related in detail and many quaint stories are told of the early professors and doctors of those days. Verily the medical men of those times did not live tamely, for we read how Robert Auchtmowtie, cherurgeane, was indicted for the slaughter of James Wauchope, with whom he had quarrelled. They fought a duel and Wauchope was killed. His relatives lodged a complaint that Auchtmowtie had set upon his adversary with two accomplices and "maist schamefullie and crewallie, with swords, straik him in the face and vpoune the heid, and gaif him four bludie woundis thairon; and thairbye maist barborouslie, crewallie and tyrannouslie slew the said vmquhile James Vauchope, vponne set purpois, provisioune and foirthocht fellonye." The Court appears to have considered it as a fair duel until King James intervened, urging diligence, and Auchtmowtie was convicted of slaughter and condemned to death. Languishing in the Tolbooth, he declared he was sick and could not bear the light. He hung one cloak outside the bars of his window and another on the inner side and "he had aqua fortis continuallie seithing at the irone window, quhill at lest the irone window wes eittine throw". Then one morning he arranged with his prentice to give him a signal by waving his handkerchief at the time when the town guard was removed, and hanging out a rope, he prepared to descend. The guard, unfortunately, had seen the signal, and so Auchtmowtie was recaptured and beheaded at the Market Cross.

An account is given of the quaint old book, "Ane breve description of the pest", which was the first medical book printed in Scotland. Written by Gilbert Skeen in 1568, it is one of the few examples of books written in the vernacular, and he gives as the main cause of "Pest in ane privat Citie is stink and corruptioun and filth, quhilks occupeis the commune streittis and gaittis".

To come to more modern times, a long list of famous medical men is passed in review. It is impossible to mention many names, so let us take one or two at random. James Gregory, whose name became familiar to us in our early youth, when at our mother's knee we were dosed with his famous powder, lives once again in these pages. Gregory was a great teacher, brilliant and witty, a great physician, a great Latin scholar, a great talker, and loved a good fight with his fellow medical men. James Hamilton, another fighter, did much for the study of midwifery, and was the last doctor in Scotland to visit his patients in a sedan chair.

The Bells (Charles, Benjamin and John), Cullen, Simpson, Lister, Argyll Robertson, Syme, Liston, Andrew Combe, to mention only a few, are all here, and the great Irish clinicians Corrigan and William Stokes, the Irish surgeon Colles were Scottish graduates.

The chapter devoted to the influence of Scottish graduates outside Scotland in the nineteenth century will be read with pride by all Scottish men. The list of great names is amazing, and when we read that such men as Pitcairn, Matthew Baillie, Richard Bright, and Thomas Addison all came from north of the Tweed, we realize the greatness of the teaching of the medical schools of the north.

Scottish graduates were instrumental in founding universities throughout the New World, for example, McGill at Montreal, and throughout Canada the influence of Scottish medical graduates was predominant.

Of interest to Australian graduates will be the portrait of Sir Thomas Anderson Stuart, and the names of the Scottish men who assisted him to build up the Sydney school are all given, with a short note of their special activities.

The volumes themselves are lavishly and handsomely illustrated with over four hundred reproductions of portraits, charters and scenes of medical interest, and the type is beautifully and clearly printed. In conclusion it may be said that here are two volumes of outstanding interest, not only to Scottish men, but to those who have not been so fortunate as to be born north of the Tweed. Those who purchase these books will often refer to them with pleasure and profit.

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INVALID PENSIONS IN AUSTRALIA.

No one will deny that the payment of invalid and old-age pensions is both necessary and wise. Especially is this true of a country that dignifies itself by the title Commonwealth. Few people, however, realize what pensions cost the State. For the year ended June 30, 1933, the sum of £10,311,626 was paid in Australia in invalid and old-age pensions. The total number of pensioners was 249,167; of these 176,425 were old-age pensioners, and 72,742 received the invalid pension. The cost of administration of the Act for the year was £92,305. Provided no hardship is inflicted on the aged or the invalid, it is in the interest of the State that the amount paid in pensions be kept as low as possible. Since invalidity can often be prevented, invalid pensions are of particular interest to medical practitioners.

In the issue of *Health* for November, 1933, invalid pensions are discussed from the viewpoint of preventive medicine. Some interesting facts are brought forward in this article. The aim and purpose of preventive medicine are quoted as "the removal of the occasion of disease and physical inefficiency, combined with the husbanding of the physical resources of the individual, in such a way and to such a degree that he can exert his full powers unhampered, at home or in the workshop, over a reasonably long life, and with benefit and satisfaction to himself and all concerned". If we look at invalid pensions in the light of this definition, we must consider how much of the illness from which invalid pensioners suffer can be prevented, and whether anything can be done for them in the way of treatment. Before a person can receive an invalid pension he must be "permanently incapacitated for work, by reason of an accident or by reason of his being an invalid". (There are other provisions, relating to age, residence in Australia, income, and so forth, that must be fulfilled.) In other words, a person who is likely to recover from his disability should not be granted a pension. In an investigation made some years ago into the causes of invalidity among pensioners under the Act, Dr. J. H. L. Cumpston found that the following conditions were most prominent: rheumatism, phthisis, disorders of the circulatory system, hemiplegia, accident, senility and congenital imbecility. He also found that 46·6% of the pensioners were under fifty and 23·7% were under forty years of age. It is, of course, obvious that much of the rheumatism, tuberculosis and circulatory and other crippling disorders in the community, caused by the streptococcus, the bacillus of tuberculosis and the spirochæte of syphilis *et cetera*, is preventible. The reduction of the number of invalid pensions occasioned by the ravages of these infective agents becomes a matter of the practice of the principles of preventive medicine. Any reduction effected in this way will be slow.

Turning to the condition of pensioners, we find an interesting state of affairs. Dr. Cumpston is reported in *Health* as having pointed out, in the investigation undertaken by him in 1916, that it was impossible to consider 6,519 persons under forty years of age as permanently incapacitated. Reference is also made to another inquiry made some years later, in which it was found that of pensioners examined physically only 38·8% among males and 50·6% among females were regarded as incapacitated to such a degree that by no means could they be rendered even partially self-supporting. In every case outside this group of 38·8% of males and 50·6% of females it was con-

sidered that the pensioner had a chance of some measure of rehabilitation in opportune circumstances. It was stressed in the report of this inquiry that it "was not intended to convey the impression that the examining medical officers had been unduly lenient". In only two instances was it thought that leniency had occurred. Vocational training to fit persons for suitable callings was indicated in 18·6% of males and 9·0% of females. Those likely to benefit by treatment comprised 31·5% of males and 33·2% of females. It must be concluded that the provisions of the *Invalid and Old-Age Pension Act* are liberally interpreted. From the humanitarian point of view no one will object. At the same time, if the adoption of this attitude is to be continued, steps should be taken to see that pensioners likely to benefit by treatment should be treated so that possibly their wage-earning capacity may be restored. Apart from the fact that the state of the public purse demands the utmost care in the administration of pension funds, it is bad for the morale of any member of the community that he should be branded as permanently incapacitated if by special treatment or training he can be made capable of earning even a small livelihood.

Current Comment.

CEREBRAL ARTERIAL DISEASE.

Perhaps the study of vascular disease may be regarded as a half-way station between the investigation of visceral disease and research in the field that must represent the goal of all biochemical and biophysical endeavour, cellular metabolism. Its importance is undoubted, for degenerative disease can largely be expressed in terms of vascular pathology. One very real difficulty is encountered at the outset in consulting current literature, that of terminology. Some authors explain what they consider is connoted by the terms they use, others do not; but agreement is by no means universal. The term arteriosclerosis offers an example; it seems to be used with increasing elasticity. In fact, one might frivolously remark that the loss of elasticity in the blood vessels parallels the gain of elasticity in the term that describes it. This is, of course, etymologically justified, but it is to be wished that writers on the subject of arterial disease would qualify this very general term so as to clarify their meaning.

C. R. Tuthill, in a recent article, describes the changes in the cerebral arteries in relation to arteriosclerosis.[1] He refers first to his own observations on the elastic layer of the cerebral vessels in children, his material being drawn from children dying of diseases not specially affecting the vessels of the brain. He found in every case raised areas of split elastic fibres at and near the branching of all the large arteries and many small ones. In the present investigation he has extended his scope to the cerebral vessels of the adult, and divides the specimens examined into seven groups; these include the apparently normal as well as different degrees of change in the raised and altered areas near the arterial bifurcations in which tissue change of varying grade is found, particularly in the elastic and collagen fibres and the lipoids. Now it may be asked if such an inquiry has any immediate bearing on clinical medicine. Undoubtedly it has, for we believe that vascular change must play an important part in many of the serious and often sudden vascular accidents that menace not only the old and outworn, but the young and active. Some of Tuthill's case histories are very interesting. For example, there is the case of the young girl of twenty-three years who fell in the street unconscious and in whom autopsy revealed a rupture of the basilar artery. No gross arterial lesion could be seen by naked eye inspection, and microscopic preparations revealed no changes in the walls of the burst vessel, and only one area at the junction of the carotid and middle cerebral arteries where the elastic tissue showed any abnormality. On the other hand, consider the case of a woman, aged forty-three, who died of a traumatic cerebellar hæmorrhage. She had been known to be a diabetic for ten years; her systolic blood pressure was 180 millimetres of mercury and the diastolic 100, yet the cerebral blood vessels only presented the appearances described by the author as occurring in young children and presumably within the bounds of normality.

It is not necessary to go into details of all the histological pictures encountered, but Tuthill's general conclusions are as follows. He thinks that it is normal to find areas of split elastic and collagen fibres near cerebral arterial bifurcations, and has evidence that these may remain unchanged during life. But where arteriosclerosis occurs, these areas increase in size and degree of fibre change. Alteration in fat occurs also; fat absorption may be noted in the hyperplastic areas, and from destruction of fat cells free lipoid may accumulate. He believes further that these changes may occur slowly or rapidly, and that they may be arrested and later recur. He cannot find a consistent ætiological factor in his series either suggested by the histological examination or by the history. Factors considered as important were hypertension, other disease, diet, blood volume, alterations in lipoid metabolism, renal disease and syphilis. He could not correlate age or physiological wear and tear with the changes observed. It must be remembered that the changes referred to

[1] *Archives of Pathology*, October, 1933.

are microscopic, for he rightly observes that only advanced changes are macroscopically visible.

It is evident from the consideration of the above that this author is referring chiefly to intimal changes of the nature of atheroma, and the confusion in terminology referred to before is to be deplored. The value of such work is, of course, unimpaired, but the difficulty is to translate into bedside terms the findings of the laboratory. The clinician must remember that he has but the crudest methods of estimating the degree and distribution of arterial disease. Definite vessel thickening can be determined, so can hypertension; study of the retinal vessels will reveal changes in the physical properties of their walls, and sometimes, as in the case of the diabetic and renal retinopathies, in their linings. But there still remain many puzzles, such as the unexplained accident detailed above. Osler's dictum about the quality of the tubing here comes to mind. But the clinician must ever realize his present limitations; he must honestly admit that he cannot always estimate the degree and type of arterial change in a given patient. A great deal of knowledge has been gained about the peripheral vessels and their behaviour in health and disease, but the all-important cerebral and cardiac arteries offer a different problem. The latter can be judged by their effect upon cardiac function, which modern methods are helping us to value, but the former still present great difficulties.

ANGINA PECTORIS.

WILLIAM EVANS AND CLIFFORD HOYLE, at the Cardiac Department of the London Hospital, have carried out a series of investigations into the efficacy of various drugs in the treatment and prevention of cardiac pain (angina of effort). Attention was recently drawn to their work in these pages. They have since recorded the results of further investigations.[1] In these they confined their attention to the treatment and prevention of individual attacks of *angina pectoris*. Patients were selected with the same care as in their former series; ninety of them were in both series. Conclusions were based largely on the patients' own experiences. The following drugs were used: glyceryl trinitrate (trinitrin), amyl nitrite, sodium nitrite, brandy, chloroform, various carminatives, and various hypnotics. Glyceryl trinitrate tablets are very apt to deteriorate; also there is the possibility that occasional tablets, owing to uneven distribution in the mass during manufacture, may contain little or none of the drug. C. A. Sykes, the pharmacist of the London Hospital, has devised a method of estimating the quantity of glyceryl trinitrate in individual tablets. The method depends on a comparison of the blue colour produced by the action of diphenylamine on glyceryl trinitrate with a standard colour obtained by the action of diphenylamine on potassium nitrate. It was revealed by the use of this test that there was little variation in the content of freshly prepared and suitably packed tablets, but that there was great deterioration after about six weeks, especially if the tablets were in open containers.

There has been considerable difference of opinion concerning the efficacy of glyceryl trinitrate; this has led to a doubt whether it is absorbed from the stomach. Evans and Hoyle show that the drug has little or no effect when swallowed rapidly, but a pronounced effect when chewed before being swallowed. When a patient with a gastrostomy opening chewed glyceryl trinitrate his pulse rate increased and his systolic and diastolic blood pressure fell rapidly; but when the drug was introduced directly into his stomach there was no change.

The practical importance of these results is that the anginal patient must chew glyceryl trinitrate tablets thoroughly to insure rapid therapeutic action from immediate absorption of the drug from the mouth. If the tablets are swallowed whole, or broken in the mouth and then swallowed quickly, they are of slight or no value in treatment.

Of 122 patients who used tablets of glyceryl trinitrate in the immediate treatment for attacks of *angina pectoris*, 105 (86%) experienced great relief, 13 moderate relief, and four no relief; objectionable effects were pronounced in two cases, slight in 28 and absent in 92. Glyceryl trinitrate in other forms was not found to be so efficacious. Tablets were made with a chocolate or lactose base with a content of glyceryl trinitrate of 0·6 milligramme (one one-hundredth of a grain) and 1·2 milligrammes. Some patients found that half of one of the smaller tablets was all that they required to obtain immediate relief; others had to take three. Tolerance did not develop, although nearly all the patients continued to take the drug for two or three years. Similar good results attended the use of glyceryl trinitrate in the immediate prevention of anginal attacks. The patients took the drug at their discretion, before exercise. Evans and Hoyle found no danger in the prevention of attacks by this means. Amyl nitrite, by inhalation, and sodium nitrite, by mouth, were not nearly so valuable in treatment and prophylaxis as glyceryl trinitrate and they had objectionable effects in a proportionately greater number of cases; amyl nitrite had severe objectionable effects in 76% and none in 5% only. The inhalation of chloroform was valueless in prevention, but relieved about half the patients to whom it was administered during attacks; it has obvious disadvantages. Brandy and carminatives are also valueless in prophylaxis; they give relief from attacks in isolated instances. For nocturnal attacks Evans and Hoyle recommend the use of glyceryl trinitrate combined with hypnotics.

An important fact made manifest by this investigation is that many patients can be so treated as to be able to take exercise that would otherwise cause them pain; apparently, within limits, this can be done without danger. Research is usually left to laboratory workers; it is refreshing to read of a scientific, well planned clinical investigation.

[1] *The Quarterly Journal of Medicine*, January, 1934.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Bronchiectasis.

W. L. ROBINSON (*The British Journal of Surgery*, October, 1933) describes a pathological study following sixteen surgical lobectomies for bronchiectasis. The outstanding and most consistent pathological finding in these series was a chronic inflammatory condition of the bronchial walls with various degrees of damage up to complete destruction of the musculo-elastic tissue. Infection of the wall would therefore seem to be of prime importance in the development of this disease. In the early stages the condition is probably of the nature of a paresis of the muscular system leading to loss of tone with consequent dilatation. Provided the infection is of short duration, recovery at this stage may be complete. When, however, the infection has persisted for a longer period of time, permanent damage occurs, the resiliency of the wall is lost, and the dilatations become fixed. With the dilatation, even if temporary, a vicious cycle is established. Bronchial secretions tend to accumulate, become infected, with spread to the adjacent walls. The damaged walls then slowly give way under the intermittent strain of ordinary or violent respiratory movements. There was nothing found in the author's series of cases to indicate that mechanical overstrain, such as pleural adhesions, collapsed lung *et cetera*, had played a part. Mechanical obstructions, such as an aspirated foreign body, tumour growth *et cetera*, are found in certain cases. None of the author's cases, however, revealed such conditions. When present, they play their part by causing a local lowering of resistance of mucosa and blocking of normal secretions. Infection then becomes established within the lumen of the bronchus, and from here soon spreads to the wall, involving the whole bronchial tree distal to the obstruction. Here it persists and leads to degenerative changes in the musculo-elastic elements of the wall. The vascular changes occurring in the bronchial arteries, if not a prime factor in the development of this disease, are no doubt an important contributory one, in that they lower the resistance of the tissues and favour persistence of the infection. In cases in which no mechanical obstruction is present such a thing as a physiological block to the clearing out of secretions might conceivably occur. A persistent local infection at the orifice of one branch may damage the ciliated epithelium to such an extent that the cilia become paralysed and the mucus in the distal portion accumulates to pathological proportions. A metaplasia of epithelium to a squamous-celled type at this point would have the same effect. Following the stagnation of secretions and infection a spread through the bronchial wall would lead to the same chain of events and terminate in bronchiectatic dilatations consequent upon damage to the muscle and elastic elements.

The Islands of Langerhans in Obesity.

ROBERTSON F. OGILVIE (*The Journal of Pathology and Bacteriology*, November, 1933) has made a *post mortem* study of nineteen cases of obesity and of nineteen control cases. In the obese group the subjects were ordinary fat people, mostly elderly women; the control subjects were persons of a lean type with little or no subcutaneous fat, but none of the controls were emaciated. The author found that in 68% of the obese subjects the islands of Langerhans manifested varying degrees of hypertrophy. He describes his method of investigation and sets out his findings in tables. He discusses the significance of his findings. He shows that in some obese subjects a hypoglycæmia is present during the occasional occurrence of symptoms similar to those resulting from an overdose of insulin. There are two possible causes of the insular hypertrophy and the state of hyperglycæmia. In the first place the consumption of large quantities of carbohydrate creates a demand for more insulin, and it may be supposed that long continued excessive carbohydrate consumption would ultimately lead to hypertrophy of the islands of Langerhans. The other possibility is that the initial change consists for some unknown reason in hypertrophy of the islands of Langerhans. The author holds that, although it is impossible to be sure wherein the initial change lies, once the carbohydrate habit is acquired and once the island tissue begins to undergo hypertrophy, a vicious circle is formed, excess carbohydrate demanding more and more insulin, and progressive hypertrophy of the island tissue creating a greater demand for carbohydrate.

Heredity and Tumour Formation.

BERNHARD FISCHER-WASELS (*Deutsche Medizinische Wochenschrft*, September 29, 1933) writes on heredity and tumour formation. He considers that for tumour development two conditions are essential—the local nucleus and the general predisposition. The local nucleus he attributes to embryonic malformation, but he points out that embryonic faults are very common and that they do not develop into tumours in the absence of a general predisposition. He has demonstrated by animal experiments that such a predisposition can be artificially produced or increased, for example, by tar poisoning of a mouse. And he quotes as of special interest the production by Büngeler of myeloid leuchæmia (which he regards as analogous to a malignant tumour) in a mouse chronically poisoned with indol. But while the cancer tendency can be thus stimulated artificially, he attaches more importance to heredity. Heredity in mice and rats has been investigated by many observers. Hill holds that three years of mouse life correspond to human old age. If mice are kept alive as long as possible they develop tumours with amazing frequency. Hill found in mice a cancer incidence of 31% for males and 49% for females—the latter higher rate being due to frequency of breast cancer in the mouse. Murray, using inbred stock, found breast tumour in 82% of the females. These researches showed that the production and the localization of both primary and secondary tumours are governed by the laws of heredity. Tumour liability and tumour resistance are inherited according to Mendel's laws. Maud Slye proved this, working with cancerous and cancer-free stock. The tendency to cancer may be transmitted through several generations without any tumour manifestation. But if two descendants of families with similar cancer tendencies are crossed, the disease appears. This is typical of many hereditary recessives. Maud Slye considers cancer tendency to be a recessive and cancer resistance to be a dominant in the Mendelian sense. Mathematical analysis by Bernstein confirms Maud Slye's theory of the recessive heredity of cancer. With the wider knowledge of the laws of heredity acquired through the animal experiments above described there is greater prospect of success in the application of eugenics. Fischer-Wasels claims that in his own clinic he has done good by opposing the union of parents unsuitable by reason of heredity. The national adoption of bolder eugenic measures will materially aid the campaign against cancer.

Pure Cholesterol Gall-Stones.

H. B. WEISER AND G. R. GRAY (*Archives of Pathology*, January, 1934) have made experimental investigations into the formation of pure cholesterol gall-stones. They give details of experiments from which they draw the following conclusions. In biliary stasis resulting from anatomical or physiological abnormalities the bile collects and becomes concentrated in the gall-bladder, where it may remain for a long period. During this period of stasis there may be an infiltration of cholesterol from hypercholesterolated blood and a decrease in the amount of alkali cholates which are responsible for retaining the fat in the form of an emulsion as well as the cholesterol in the dispersed state. In the absence of infection, a decrease in the alkali cholates may result from either of the following causes: (1) a change in the pH of the bile from the alkaline to the acid side, thereby converting the alkali salt to the insoluble glycocholic acid, which is neither an emulsifying

agent for fat nor a peptonizing agent for cholesterol; or (ii) a physiological change in the wall of the gall-bladder, which allows resorption of the alkali cholates. The author thinks that there is no doubt that the first of these factors will contribute to the precipitation of cholesterol, since bile from the gall-bladder is normally acid, while that from the hepatic duct is alkaline. This normal change in pH value is probably accentuated in biliary stasis. In the normal gall-bladder there is little or no resorption of alkali cholates. Any resorption of cholates that leads to the formation of a pure cholesterol stone must result from a physiological derangement that does not produce histological changes. Clinical irritation that is not sufficiently severe to leave a permanent change in the tissue may be a contributing factor. The disappearance of alkali cholate, either by conversion to glycocholic acid or by resorption, causes precipitation of cholesterol. The excess cholesterol collects around the fat droplets which tend to coalesce as the cholate is gradually removed. Clumps of fat interspersed with cholesterol result, and the process of solution of the finely divided particles and of subsequent reprecipitation in large needle-like crystals binds the mass together. The continuation of this process for a long period leads eventually to the concrement which consists of relatively large crystals of cholesterol together with a small amount of enclosed fat.

MORPHOLOGY.

Sympathetic Cells in the Spinal Ganglia.

CHARLES FISHER AND S. W. RANSON (*Journal of Anatomy*, October, 1933) discuss the work of Kiss, who has recently reported a series of experiments on the cranial and spinal ganglia of man and different mammals, in which he claims to have demonstrated that sensory and sympathetic nerves meet in the same ganglion. As the results of these experiments, if confirmed, would lead to a considerable revision of our present conception of the anatomy of the nervous system, the present authors repeated Kiss's work and attempted to find an explanation of his findings. After prolonged treatment of the tissues with osmic acid following preliminary fixation in formalin, Kiss was able to differentiate two types of cells which he claims are sensory and sympathetic respectively. The latter appear as black cells and usually are smaller than the former. The present authors conclude that the technique employed causes a small proportion of the ganglion cells to become black, while others are lightly stained and still others take various shades of grey. The depth of the stain seems to be related to the amount of shrinkage. If before fixation the ganglia are treated with hypertonic saline solution or are crushed, the number of shrunken dark cells is increased. Delay in fixation, allowing *post mortem* changes to occur in the ganglia, also increases the number of dark shrunken cells. No evidence was found in support of the theory, proposed by Kiss, that the dark cells in the spinal ganglia are sympathetic.

The Rôle of Vestibular Nuclei in the Cortical Innervation of the Eye Muscles.

E. A. SPIEGEL (*Archives of Neurology and Psychiatry*, May, 1933) finds that bilateral injuries to the vestibular nuclei change the direction of eye movements elicited by stimulation of the occipital as well as of the frontal lobe. Conjugate deviation of the eyes to the side opposite the stimulated cortical area, in particular, depends on the intactness of the vestibular nuclei. After exposure of the floor of the fourth ventricle, stimulation of the occipital lobe produced conjugate lateral ocular movement to the opposite side in 54·1% and vertical deviation of the eyes in 5·5% of the author's experiments. After bilateral lesions of the vestibular nuclei were produced, stimulation of the occipital lobe produced lateral deviation of the opposite side in 3·4% and vertical deviation in 48·2% of the experiments. The experimental findings are explained by the assumption that the vestibular nuclei play an important rôle in the cortical control of the muscles of the eyes for lateral movements. The neurones of the vestibular nuclei seem to carry impulses not only from the labyrinth, but also from the frontal and occipital lobe into the posterior longitudinal fasciculus.

The Structure of the Sinuses in Lymph Glands.

C. K. DRINKER and others (*The Anatomical Record*, June, 1933) injected with Indian ink lymph glands of young adult dogs and state that the walls of the sinuses in the injected glands are formed by reticulo-endothelial cells, not by lining endothelium similar to that in the afferent and efferent vessels of the glands. They also state that the sinus walls are incomplete wherever lymphocytic growth is active, and lymph passing along the sinuses runs for varying distances into the lymphocytic collections.

Intrinsic Fibres of the Cerebellum.

JAN JANSEN (*Journal of Comparative Neurology*, June, 1933) describes an experimental investigation of the rabbit's cerebellum and subsequent examination of the tissue by the method of Marchi. He finds that all folia of the cerebellar cortex are connected with the neighbouring folia (two or three on both sides) by arcuate fibres. Association fibres are found linking up the parts within the floccular formation, but no associational connexions exist between the cortex of the floccular formation and the cortex of either hemisphere or vermis. There are no associational tracts from the vermis to either hemisphere or floccular formation. Associational fibres connect the cortex of the hemisphere with the vermis. The hemispheres are interconnected by association fibres which, partly at least, seem to have the character of a commissure. Finally, the study of the association fibres in general confirms the impression of mutual independence of the main cerebellar lobes.

The Development of the Hypophysio-Portal System.

PAUL G. 'ESPINASSE (*Journal of Anatomy*, October, 1933) gives an account of the development of the so-called hypophysio-portal veins, which were recently described by Popa and Fielding. He concludes that the hypophysio-portal vessels are a part of the arterial supply of the brain. This part of the arterial supply has been secondarily involved and enclosed by the upgrowing wings of the buccal part of the pituitary when it was close to the under-surface of the brain. As the infundibular stalk lengthens, the vessels assume their distinctive form.

The Normal Histology of the Intradural Filum Terminale.

J. W. HARMEIER (*Archives of Neurology and Psychiatry*, February, 1933) reviews the literature of the histology of the intradural *filum terminale* and refers to its importance because of the formation of tumours in this region. He finds that the *filum terminale internum* contains all the elements found in the spinal cord; the more distal the segment examined, the less neurogenic tissue is found and the greater is the decrease in the component parts of the neurogenic tissue. A large vein and two small arteries are found in association with the neurogenic tissue. The author concludes that tumours of this region therefore should be similar in type to those found in the spinal cord.

The Muscle Content of the Lower Part of the Œsophagus of Man.

L. B. AREY AND M. J. TREMAINE (*The Anatomical Record*, July, 1933) examined microscopically the lower thirds of seventy-four human œsophagi at four different levels for skeletal muscle fibres. Only one individual (a negro) contained any skeletal muscle at these lower œsophageal levels. This striate muscle content occurred as fascicles which amounted to 63% of the longitudinal muscle coat and 20% of the circular coat. It is possible that a continuation of skeletal muscle fibres on to the stomach wall occurred. Contrary to certain statements which have been made from time to time, the occurrence of skeletal muscle in the lower part of the œsophagus in man must be considered anomalous.

Special Articles on Treatment.

(Contributed by request.)

XXVIII.

THE TREATMENT OF CHOLECYSTITIS.

The successful treatment of cholecystitis depends largely upon the ætiology, pathology and complications of this condition. Therefore a discussion of therapeutic measures must be preceded by a brief account of the morbid condition, its antecedents and accompaniments.

Cholecystitis exists in two main forms: (*a*) acute, (*b*) chronic.

Acute Cholecystitis.

The acute form may occur:

1. As a complication of acute febrile infection, of which the most important are enteric fever and the paratyphoids. In enteric fever cholecystitis may occur: (*a*) as a catarrhal inflammation brought about by infection of the bile by *Bacillus typhosus*, from hepatic foci; and (*b*) from the occurrence of typhoid ulcers beginning in the cholecystic submucosal lymphoid tissue. I have seen two cases of perforation of the gall-bladder in enteric fever due to deep necrosis of such ulcers.

2. Acute exacerbations of inflammation may occur in a gall-bladder which is already the site of chronic cholecystitis. In these cases, usually, but not always, gall-stones are present.

3. Biliary stasis undoubtedly predisposes to attacks of acute cholecystitis. Stagnation of bile may be brought about by obstruction to its flow, usually from calculus near the neck of the gall-bladder or from inflammatory infiltration in the walls of the gall-bladder, which renders them more or less immobile. Adhesions between the serous coat and surrounding structures may interfere with normal emptying movements.

4. In cases of deranged gastric and duodenal digestive function acute cholecystitis may occur. Thus it is a not infrequent complication of gastric achlorhydria or achylia.

From the foregoing brief, and by no means exhaustive, *résumé* of the causes and accompaniments of acute cholecystitis, it is apparent that the treatment of an individual case largely depends upon these factors.

(*a*) Cholecystitis during the course of an acute fever calls for special measures for the relief of pain and for the alertness as to the possibility of sudden perforation (which is rare). For the pain, hot, moist applications may be used. In this respect cataplasms are inadvisable, since they hinder careful examination of the affected region. Hypodermic injection of morphine sulphate, 0·01 gramme (one-sixth of a grain), with atropine sulphate, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), may be necessary, but should not be frequently repeated lest danger signals be masked. Usually such an attack lasts for a few days and then subsides.

(*b*) When acute cholecystitis occurs as an exacerbation of a chronic case, the treatment is similar to the above, except that aspirin may be freely used and morphine is seldom necessary. If the acute cholecystitis is but a part of an acute infective cholangitis, then surgical intervention is almost certainly advisable.

As this article is concerned with medical treatment, the nature of the operation to be performed will not be discussed, but the indications for operation are: (i) manifestations of increasing sepsis (dry tongue, exhaustion, high fever with rigors); (ii) increasing jaundice. Vomiting may be persistent and urinary secretion much diminished.

In such a case, if operation is decided upon, very careful medical preparation of the patient is necessary because of his poisoned condition, the impairment of hepatic function, and the probably lowered coagulability of the blood. The careful choice of anæsthetic, the administration of large amounts of glucose, and possibly "Hemostatic serum" must be considered.

(*c*) In cases of biliary stasis due to partially obstructed outflow with acute inflammation of the gall-bladder, resolution may be assisted by measures such as those outlined in Section (*a*), but (i) increasing distension of the gall-bladder, or (ii) high fever, increasing pain, and leucocytosis due to empyema may necessitate cholecystotomy. At the same time the surgeon may be able to remove the cause of the obstruction or he may decide to perform cholecystectomy.

5. The association of acute cholecystitis with achlorhydria is an interesting one. Conceivably it may be due either to failure of efficient emptying of the gall-bladder owing to the lack of stimulus normally afforded by the acid gastric juice entering the duodenum, or it may be that the protective disinfecting power of the gastric juice is diminished and the gall-bladder suffers *viâ* the contaminated portal blood → liver foci → bile sequence, as in enteric fever. In any case, after treating the acute condition, it is necessary, as far as possible, to promote normal gastric digestion, by the administration of dilute hydrochloric acid, four cubic centimetres (one drachm), well diluted with water, with and after food.

Chronic Cholecystitis.

Chronic cholecystitis offers many problems in treatment. Both the ætiology and pathology of this condition are obscure, although the morbid anatomy is well known.

Chronic cholecystitis occurs most commonly as a sequal to an acute attack or as an accompaniment of gall-stones, or associated with chronic appendicitis. The infective agent reaches the gall-bladder by the blood stream, rather than by the lymphatics or mucous membrane. All the coats of the gall-bladder may be affected in varying degree, and the symptoms and sequelæ are to some degree due to the site of the main incidence of the inflammation.

The sufferer from this disease is usually a middle-aged, fat, parous woman of a dark "bilious" complexion, who complains of flatulent dyspepsia, and sometimes of dull pain below the right rib margin and/or beneath the right shoulder blade. In addition, there may be a history of pain due to biliary colic or of attacks of jaundice. The symptoms have been well described by Moynihan.

In the management of such a case, presuming that the diagnosis of chronic cholecystitis is established, it is important to determine: (*a*) the condition of the gall-bladder anatomically and functionally, (*b*) whether calculi are present.

If facilities for its use are available, cholecystography will usually prove of great assistance, but in the event of the first examination failing to show the gall-bladder, the test should be repeated.

Sometimes duodenal intubation may be of value in revealing pus and the nature of the infective agent. Occasionally the latter proves to be the typhoid bacillus.

If the history, examination and special investigations indicate the presence of gall-stones as well as of cholecystitis, medical treatment is unlikely to be more than palliative, and if the condition of the patient justifies it, operation should be advised.

If, however, there is good reason to believe that gall-stones are not present and that the gall-bladder is not permanently damaged, then medical treatment may be adopted. This must also be done in cases in which operation is for some reason inadvisable, or when the patient refuses operation.

The chief indications for treatment are: (*a*) stimulation of contraction of the muscular wall of the gall-bladder, with consequent emptying, (*b*) avoidance of acute inflammation, (*c*) disinfection of the bile.

In my experience the first indication can often be fulfilled, the second sometimes, and the third very seldom.

Radiology has shown that the normal gall-bladder contracts and empties itself if fat enters the duodenum, and reacts in the same way to magnesium sulphate or sodium sulphate.

For many years olive oil taken on an empty stomach has been used as a remedy for gall-stones, and in successful cases has probably acted by its stimulation of the emptying reflex of the gall-bladder.

The drugs usually employed are magnesium sulphate or sodium sulphate, one teaspoonful dissolved in about one tablespoonful of water. The dose should be taken in the morning fasting, and no food or drink should be consumed until an hour afterwards. This will induce a satisfactory movement of the bowels as well as assisting to drain the gall-bladder.

As these patients are usually over fat, the diet should be appropriately regulated and gentle exercise and perhaps massage recommended. It is well to examine the heart and circulation carefully in such patients, for, as Cabot has pointed out, myocardial weakness is very common amongst patients with chronic gall-bladder disease.

A large quantity of fluid should be consumed. This is advisable since it stimulates both intestinal peristalsis and biliary secretion.

These methods may be sufficient to improve the patient's condition gradually and possibly to lead to resolution of the morbid process.

Duodenal lavage with Einhorn's tube has its advocates. Whether it is possible to disinfect the bile once it is the nidus of vigorous bacterial growth is, I believe, very doubtful. For many years sodium salicylate has been recommended and used for this purpose. Recently A. F. Hurst has advocated large doses of hexamine with alkalis.

Some time ago I had a patient who discharged bile infected with *Bacillus typhosus* through a cholecystostomy opening. The number of bacilli per cubic millimetre of bile could be counted. We administered several so-called biliary disinfectants, but the count was not affected by any of them.

Doubtless other disturbances of the alimentary tract will aggravate chronic cholecystitis, and they should be sought for and corrected. The two most frequently encountered are gastric achlorhydria and chronic appendiceal disease.

Finally, it should be remembered that chronic gall-bladder disease is definitely a precursor of cancer of the gall-bladder, which occurs in a relatively high proportion of such cases. Therefore, if the patient does not respond within a few weeks of medical treatment, the question of operation should be seriously considered, even if gall-stones are absent.

C. T. Ch. de Crespigny,
M.D., F.R.C.P. (London),
Lecturer in Principles and Practice of Medicine in the University of Adelaide; Physician, The Adelaide Hospital; Consulting Physician, Children's Hospital, Adelaide.

British Medical Association News.

SCIENTIFIC.

A meeting of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on February 2, 1934, Dr. W. N. Robertson in the chair.

Chronic Indigestion.

Dr. Alan E. Lee read a paper entitled: "Chronic Indigestion" (see page 395).

Dr. R. Graham Brown said that as Dr. Lee's paper was of such a high standard there was evidently some hesitation on the part of the audience to discuss it. He felt that he would set the ball rolling by starting it. It seemed rather absurd that such a discussion should be opened by a surgeon whose province was the head and neck. Speaking for himself, he said that he had derived great pleasure and much enlightenment in listening to the excellent exposition that Dr. Lee had given.

He asked for some explanation of the symptom of flatulence which was so commonly met with, for he could not believe that all flatulence was due to swallowed air, and he instanced the case of a personal friend who immediately after drinking a glass of milk suffered from wind, although other beverages and food did not produce this symptom.

Regarding the relationship of infection about the mouth and throat to disorders of the alimentary tract, he considered that it would be a good policy for all foci of infection to be removed from those regions before any operation upon the upper part of the abdomen and the stomach in particular was performed. He quoted instances of patients that had come under his care upon whom gastro-enterostomy had been performed, in certain instances by prominent gastric surgeons in Great Britain. No relief had been obtained from the pain after the abdominal operation, whereas within a short period after removal of septic tonsils patients had on several occasions obtained complete relief. Sepsis in the mouth and throat accounted for a great many gastric symptoms, and he remarked that he knew of a number of patients upon whom big gastric operations had been performed where this factor had not been attended to, with consequent unsatisfactory results as far as relief of the original abdominal symptoms was concerned.

Dr. J. Lockhart Gibson congratulated Dr. Lee on his paper. It was most gratifying to find a surgeon insisting that help from medical treatment should be exhausted before operative treatment in cases of chronic indigestion was considered.

Dr. Lee had mentioned more than once the sensitiveness of the cornea. He (Dr. Gibson) was interested in a possible parallel between the referred pain in gastric ulcer and so-called corneal pain. The cornea was, of course, very sensitive, but in corneal lesions, for instance in phlyctenular keratitis, the pain felt by the patient was due to irritative effect on the sphincter of the iris. In such cases the instillation of atropine gave relief, and did so very quickly. When the pupil had become fully dilated and was kept dilated, pain in the eye from corneal wounds or ulcers practically ceased.

He did not think that physicians, surgeons and neurologists recognized sufficiently the effect on many conditions of uncorrected errors of refraction. It was the small errors that were such important factors in inducing and increasing nervous symptoms. Their connexion in susceptible people had often a dramatic effect, when much other treatment had failed.

Dr. E. S. Meyers congratulated Dr. Lee on his excellent paper. With regard to the point raised by Dr. Brown as to sepsis in the mouth, Dr. Meyers thought that the general surgeon had great difficulty in realizing when patients had that type of sepsis, especially tonsillar trouble. He would like to see the ear, nose and throat specialists lay down some definite standard for this.

When speaking of chronic ulcer, Dr. Lee had said that sepsis did not play the important part it was supposed to play, but in acute ulceration sepsis played a part. From the literature he had read Dr. Meyers had thought it was otherwise. Dr. Meyers agreed with Dr. Lee in all his conclusions about visceroptotic patients. He considered his arguments sound and said that it must be recognized that these cases did occur and needed treatment. Dr. Lee had shown the great difficulty there was in diagnosing these cases; Dr. Meyers's difficulty as an in-patient surgeon had been in stopping the out-patient surgeons and physicians from sending such patients in for operation. Dr. Meyers often had to send home patients thus referred, whom he did not consider needed operation. Of course the clinics were very large and surgeons were rushed. Dr. Meyers often sent these patients to the massage department for exercises *et cetera*. He wondered if something else could not be done; there were tuberculosis clinics, allergy and diabetic clinics. Could there not be a clinic where these patients could have more attention and their problems be solved. In other countries there were systems where patients had definite appointments and time was set aside for each case, then these could

be sorted out and not so much surgical interference would take place. The theory as to the mechanism of pain set forth by Dr. Lee had been taught in Dr. Meyers's time at Sydney University by Professor Mills, Lecturer in Medicine, and was known as Mackenzie's theory. Dr. Lee deserved well for all the trouble he had taken.

DR. JOHN BOSTOCK congratulated Dr. Lee on his presentation of the subject. He considered that a third mechanism should be considered in the production of nervous dyspepsias. Numerous people had a sensitive recording apparatus which allowed normal visceral stimuli to reach consciousness. This type of individual became too easily aware of peristaltic changes which were neither exaggerated nor morbid.

Dr. Lee, in reply to Dr. Graham Brown, said that the problem of belching was a difficult one which he did not entirely understand. While he thought air swallowing was the most important factor in it, some reversed peristalsis mechanism might well initiate the attacks. The difficulty about bringing reversed peristalsis into the picture was that it was well established that these waves in the stomach elicited a subjective sensation of nausea which was not necessarily present in these patients.

Regurgitation of duodenal contents was a normal condition as well as a condition that the surgeon frequently encouraged by the operation of pyloroplasty. Such regurgitation produced bicarbonates and not carbon dioxide.

Dr. Lee expressed his interest in Dr. Gibson's statements about the sensitivity of the cornea. In reply to Dr. Meyers, Dr. Lee said he could produce much current literature confirming the relationship of acute ulceration to sepsis and of the experimental production of chronic peptic ulcer by acid alone.

Dr. Meyers had maintained that members of the honorary staff of the Brisbane Hospital, including Dr. Lee, appeared too eager to subject patients to the operation of colopexy. The reply to this was that the treatment of patients in private practice and in hospital practice must frequently diverge. As a matter of expediency it was often better to subject the working man to an operation that would get him back to a gainful occupation speedily than to carry out a long course of medical treatment.

Much more could be done in the organizing of gymnastic classes for both hospital and private patients, but it was doubtful whether the massage department could carry on such further activities at present.

In reply to Dr. Bostock, Dr. Lee said that he did not presume to discuss the factors behind a nervous dyspepsia, but only suggested the mechanism by which nervous derangements could influence the intestinal tract.

He agreed that a heightening of sensitivity on the afferent side was an undoubted factor in the production of these patients' complaints.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Sydney Hospital, Sydney, on October 19, 1933. The meeting took the form of a series of demonstrations by members of the honorary staff.

Auricular Fibrillation.

DR. WILFRED EVANS showed two patients who had suffered from auricular fibrillation and had been treated with quinidine.

The first was a male, aged forty-four years, who had suffered from breathlessness since January, 1933, but no pain. There was no history of rheumatism. His apex beat was found to be in the fifth intercostal space inside the nipple line. The area of cardiac dulness was not enlarged. The cardiac rhythm was grossly irregular and a loud systolic murmur was heard at the apex. No ascites or liver enlargement was found. No abnormality was found in the urine. X ray examination revealed enlargement of both right and left ventricles. Electrocardiographic examination revealed auricular fibrillation. The Wassermann reaction was completely positive.

The patient was given digitalis until his pulse rate was reduced to 60 per minute, and after two days quinidine was given, in a dose of 0·3 gramme (five grains) a day, then in increasing doses to 1·8 grammes (30 grains) a day for three days. The condition of impure auricular flutter then developed and quinidine administration was suspended. Tincture of digitalis was then given in doses of eight cubic centimetres (two fluid drachms) a day for ten days; a further course of quinidine was then given, and normal rhythm was restored.

Dr. Evans remarked that the interesting feature was that the patient had no previous history of rheumatism but reacted to the Wassermann test. The question arose whether his syphilitic infection was a factor in the production of the auricular fibrillation.

The next patient was a woman, aged thirty-eight years, who had been admitted to hospital on March 20, 1933. She had rheumatism at twenty-one years of age and was quite well until her first confinement in July, 1932, when she developed marked shortness of breath, which had persisted ever since and recently had become much worse. The apex beat was in the fifth intercostal space, 12·5 centimetres (five inches) to the left of the middle line. The cardiac rhythm was grossly irregular and a late diastolic murmur was heard in the mitral area. Her pulse rate was 160 per minute. She was definitely distressed, with cyanosis of the lips and hyperpnœa. The diagnosis was auricular fibrillation. There was no reaction to the Wassermann test.

Tincture of digitalis was administered in doses of two cubic centimetres (30 minims) three times a day until April 1, 1933. On April 4, 1933, quinidine in a dose of 0·3 gramme (five grains) was given at night, and the next day three doses of the same size were given; the dose was increased by 0·3 gramme (five grains) until 1·8 grammes (30 grains) a day were given; this dose was continued for three days. Normal rhythm was then restored, and this was shown in the electrocardiographic tracings.

The patient was about four months pregnant at that time and it was decided that if pregnancy were allowed to continue she might lose her life, so miscarriage was induced on May 27, 1933. The patient's condition was good and her rhythm remained normal. Sterilization had since been carried out.

Heart Block.

Dr. Evans also showed a man, aged sixty years, who complained of breathlessness on exertion. Physical examination revealed the classical signs of complete heart block. There was no marked cardiac enlargement, but a loud systolic murmur was heard at the apex. The systolic blood pressure was 150 and the diastolic 50 millimetres of mercury. There was no reaction to the Wassermann test.

The X ray report was as follows: "Heart not enlarged; enlargement of bronchial glands; pleural adhesions both bases."

Electrocardiographic examination revealed complete heart block. The ventricular rate was 30 per minute and the auricular rate 110 per minute. The rhythm was regular. The *P* waves were upright throughout; they were completely dissociated from the ventricular rhythm. The *Q R S* complexes were upright in Leads I and II and inverted in Lead III; they were very notched and widened to 0·12 second. The *T* waves were inverted throughout.

Dr. Evans remarked that in 1931, for a period of some months, the signs of heart block clinically and, as shown by the electrocardiograph, completely had disappeared.

Secondary Syphilis Associated with Myocarditis.

Dr. Evans's next patient was a man, aged twenty-nine years, who had suffered from œdema of the ankles for a fortnight prior to admission. He had no breathlessness or pain. He was a barman and had drunk a great deal of alcohol. His systolic blood pressure was 135 and diastolic 70 millimetres of mercury. The apex beat was in the sixth intercostal space in the anterior axillary line.

The area of cardiac dulness was greatly increased to the left. There was marked gallop rhythm, but no murmur. The tone of the heart sounds was not good. The liver was enlarged and hard. The urine contained no albumin or sugar. In the electrocardiogram the *T* waves were poorly developed and there was evidence of sinus tachycardia. X ray examination revealed great ventricular hypertrophy. The result of the urea concentration test was normal. The Wassermann reaction was strongly positive.

Dr. Evans remarked that this was an extremely interesting patient exhibiting marked cardiac enlargement and signs of failure, and the only ætiological factor that could be found was the syphilitic infection.

Treatment consisted in the administration of mercury, iodides and "Salvarsan".

Aortic Regurgitation.

Dr. Evans also showed a male patient who had suffered from breathlessness on exertion since July, 1930. The apex beat was in the fifth intercostal space, 12·5 centimetres (five inches) from the middle line. The heart sounds were of poor tone; the rhythm was of the pendulum type. The systolic blood pressure was 160 and the diastolic 95 millimetres of mercury. There was evidence of severe emphysema. X ray examination revealed "marked cardiac enlargement with diffuse aortic dilatation". The Wassermann reaction was completely positive. Electrocardiographic examination revealed that the cardiac rate was 110 per minute and the rhythm was regular. The *P* waves were upright in Leads I and II and inverted in Lead III. The *P-R* interval was 0·16 second. The *Q R S* complexes were upright in Leads I and II and diphasic in Lead III. The *T* waves were upright in Leads I and II and deeply inverted in Lead III. The only feature suggestive of disease was the very deep inversion of the *T* wave in Lead III. Treatment consisted in the administration of a full course of "Salvarsan" and mercury after preliminary treatment with iodides by mouth and mercurial inunction.

Aortic Stenosis.

Dr. Evans next showed a woman, aged eighty-one years, who complained of breathlessness on exertion. She had an enlarged left ventricle, and there was a rough systolic thrill in the aortic region. The systolic blood pressure was 145 and the diastolic 120 millimetres of mercury. The wall of the radial artery was thickened. A loud systolic murmur and a very much diminished second sound could be heard in the aortic region. Numerous extrasystoles were present. X ray examination revealed great ventricular hypertrophy and diffuse aortic dilatation. Electrocardiographic examination revealed numerous extrasystoles, left preponderance and early heart block.

Microcytic Anæmia.

Dr. Evans also showed a woman, aged fifty-two years, who had suffered from dizziness and shortness of breath for some years. She was anæmic in appearance and had a typical achlorhydric tongue. Atrophic changes were to be seen in the finger nails. The systolic blood pressure was 170 and the diastolic 100 millimetres of mercury.

Fractional test meal examination revealed complete achlorhydria. The red blood cells numbered 4,830,000 per cubic millimetre; the hæmoglobin value was 55%; the colour index was 0·58; the leucocytes numbered 6,720 per cubic millimetre, and of these there were 68% polymorphonuclear cells, 2% eosinophile cells, and 30% lymphocytes. The red cells were irregular in size; but the majority were small, and microcytes were numerous. No macrocytes were seen. There was poikilocytosis, and the cells were poorly filled with hæmoglobin, many being mere rings of cytoplasm. Polychromasia was present, but not marked. No nucleated forms were seen.

Skiagrams.

DR. J. G. and DR. W. A. EDWARDS showed a series of skiagrams.

A skiagram of lungs affected with early silicosis and one of lungs affected with miliary tuberculosis were shown. It was pointed out that it was impossible to differentiate radiographically, without the clinical history, between the two.

The radiographic appearance of hydropneumothorax was demonstrated in another film, with irregular shadows over the neck and thorax due to an escape of lipiodol into the areolar tissues.

Radiograms of diverticula of the upper and lower ends of the œsophagus, of the stomach and duodenum, and of the colon, filled with barium sulphate, were shown.

Three skiagrams of hour-glass stomach due to a band ulcer and malignancy respectively were exhibited, also of a calcifying hydatid pressing on the stomach and one pressing on the gall-bladder, which was filled with "Opacol".

In another skiagram was shown the appearance presented by a Riedel's lobe of the liver, which could be felt clinically below the right costal margin.

Numerous skiagrams greatly reduced in size, depicting various pathological conditions of the alimentary tract and of the cranial sinuses, were shown.

Pan-Sinusitis.

DR. H. S. MARSH showed a female patient, aged twenty years, who had been admitted to the eye department on February 26, 1933. She had been well until three days before admission, when she had had nausea, vomiting, a stinging sensation in the right eye, blockage of both nostrils, and nasal discharge. Next morning there had been great swelling of the right eye. At the time of admission examination revealed great swelling of both eyelids, extending across the forehead. There was proptosis of the right eye; the eye movements were impaired. The cornea was normal. There was chemosis. The temperature was 39·5° C. (103·2° F.), and the pulse rate 110 per minute. The urine contained albumin.

The right orbit was explored, but no pus was found. X ray examination two days later revealed opacity of the frontal, ethmoid and right maxillary sinuses. The leucocytes numbered 19,600 per cubic millimetre of blood. The patient was then transferred to the ear, nose and throat department. By this time the eyeball was almost filled with œdematous conjunctiva; the right side of the face was very swollen and œdematous. Pus was oozing from the right nostril; the mucous membrane on the right side was inflamed and œdematous; the left side was in a similar condition, but was much less severely affected. Under ether anæsthesia an incision was made in the canine fossa. Pus oozed down from above when the periosteum was being stripped. The right maxillary antrum was full of thick creamy pus; this was evacuated and the antrum packed with gauze; the mucous membrane was not removed. Incision was then made over the right frontal sinus, carried round the inner canthus in the usual manner; the sinus was opened and found full of pus; the outer and inferior walls were totally removed. The upper part of the nasal process of the maxilla was removed also. A large subperiosteal abscess was found connected with the right ethmoid. The right ethmoid body was then removed and found full of pus and broken-down bone. The cavity was packed. A similar operation was then performed on the left side, excepting that the left antrum was not opened. No pus was found in the orbit on either side. The wounds were packed twice a day until they granulated up. A plastic operation was used to close the wounds. Later it was found necessary to remove the mucous membrane and muco-periosteum of the right antrum. Dr. North also removed some redundant conjunctiva. The result was cure with the loss of sight of the right eye.

Encephalomyelitis.

DR. G. C. WILLCOCKS showed a man, aged thirty-six years, who had had a chronic infection of the sinuses with offensive purulent discharge, accompanied by asthma, for two years. Some months before the meeting he had noticed loss of power in both legs of gradual onset, accompanied by weakness of the arms. At the same time he

had a severe cough, with abundant purulent sputum. When first seen, in addition to these disorders, there were definite clinical and radiological signs of abscess of the lung. The patient lay on his back with his arms flexed at the elbows, with marked wrist-drop on both sides, in the characteristic posture of a patient with a fracture-dislocation of the spine at the level of the fifth or sixth cervical vertebra. There was loss of power in all muscles of the arms, less marked in the biceps and flexors of the wrist. There was marked paresis of all muscles of the legs; dorsiflexion of the foot was absent. All reflexes of the limbs were absent. The sphincters were unaffected. The abdominal reflexes were not obtained. Sensory changes were indefinite. The sight was markedly affected in both eyes, the right eye being practically blind. The sinuses were treated, with much improvement. The lung abscess subsided. The nervous signs altered little, except that sight improved; at the time of the meeting there was evidence of optic atrophy with a degree of hemianopia, and the extensors of the right wrist had recovered power. There was diminution of sensation over the distribution of the eighth cervical and first dorsal segments.

Dr. Willcocks remarked that the original diagnosis had been encephalomyelitis causing optic neuritis and atrophy, with the main spinal cord lesion at the fifth and sixth cervical segments; there was now evidence that the lesion had also involved the seventh and eighth cervical and first thoracic segments. The affection of the legs indicated a marked involvement of the lumbar segments of the cord. Multiple peripheral neuritis was unlikely, in view of the optic atrophy and the lack of recovery in power, as well as the segmental distribution of sensory loss. The gross sinus infection may have predisposed to myelitis.

Pneumonokoniosis or Tuberculosis.

Dr. Willcocks showed a male patient, aged sixty-six years, who stated that he was short of breath, and complained of a variety of symptoms, of which cough was one. The only definite chest signs, apart from those of age, were the presence of rhonchi. At the time of the first examination he had influenza. He had been engaged in coal and gold mining all his life. The X ray report was as follows:

> In the left lung there is a considerable increase in mottling of a rather stippled type, with some enlargement of the left hilar shadow. In the right lung there is a considerable increase in dulness, extending downwards with what appear to be some early cavities. In addition, there are adhesions at both bases, especially the right. The appearance is not typical of pneumonokoniosis, but suggests rather an old chronic tuberculous infection of the left lung with an inflammatory lesion of the right lung, possibly following an old interlobar pleurisy or empyema, possibly a post-influenzal condition. Should like to make another examination, taking a stereoscopic and a lateral picture.

Dr. Willcocks remarked that, presuming the mottling at the base of the right lung to be due to chronic and not a recent acute condition, it was interesting to speculate on what part his work had played in causing his present disablement.

Dr. Willcocks's next patient was a man, aged seventy-two years, who had complained of tightness in the epigastrium and cough with white sputum, sometimes blood-stained, for two years; he had been short of breath for the same period. He was an old age pensioner. He stated that he had been a gold miner all his life. Most of his work had been done on the surface. Apart from the signs of his age, including an aortic systolic murmur, there was no outstanding physical sign. The X ray report was as follows:

> Definite increase in mottling in both lungs, confined mainly to the apices and middle thirds. Appearance suggests tuberculosis, the lesion involving both upper lobes with extension downwards. It is difficult to exclude the possibility of pneumonokoniosis.

The sputum was examined three times for tubercle bacilli, but they were not found.

Dr. Willcocks remarked that the case illustrated the difficulty of determining what part silicosis might play in causing the disablement of old miners. Silicosis would not be expected to develop to any extent in a surface miner; and in any case it would not seem to have shortened this man's life by many years.

Subacute Combined Degeneration.

Dr. Willcocks's last patient was a man, aged forty-two years, who had suffered from numbness of the legs and hands for sixteen months. No evidence of organic disease had been found, except absence of the ankle jerks. There was a deficiency of hydrochloric acid in the gastric contents. There was no marked improvement as a result of the administration of liver and hydrochloric acid. There was diminished vibration sense in the left ankle and leg. The *tendo Achillis* was insensitive. The patient swayed, and he walked clumsily. There was slight Rombergism.

Dr. Willcocks said that the diagnosis was between *tabes dorsalis* and subacute combined degeneration of the cord.

(*To be continued.*)

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Quilter, John William, M.B., B.S., 1930 (Univ. Sydney), Grenfell, New South Wales.

Medical Practice.

POLIOMYELITIS IN SYDNEY.

THE Infantile Paralysis Committee of New South Wales wishes to draw the attention of medical practitioners to the fact that several sporadic cases of anterior poliomyelitis have occurred recently in Sydney.

The medical officer of the Committee is prepared to assist in the early diagnosis by doing a lumbar puncture and examining the cerebro-spinal fluid and will administer serum in suitable cases. Symptoms which are commonly found at the onset are pain in the head or back, pain on anterior flexion of the neck or back, together with fever. The patient's facial expression is often anxious and apprehensive. This is a characteristic feature of the early phase of the disease.

The telephone number of the Infantile Paralysis Committee, Sydney, is B 1537.

Correspondence.

TUBERCULIN.

SIR: On reading the proposed syllabus for the post-graduate course in tuberculosis one is immediately struck by the omission of any reference to tuberculin in diagnosis or treatment, unless one can call "skin tests" (by which is presumably meant the von Pirquet and Mantoux tests) such a reference.

It is worth noting that at the Centenary Congress of the British Medical Association in London in 1932 special sessions were devoted to a discussion on "The Position Today of Tuberculin in Diagnosis" and "The Position Today of Tuberculin in Treatment". During those discussions the majority of the speakers admitted that tuberculin had a definite use both in diagnosis and in treatment. The minority who condemned it whole-heartedly appeared to do so without any scientific reason—possibly an inherent dislike of it—for it is quite certain they had not given it a scientific trial on approved lines.

Further, at the recent congress in Hobart, as reported in the journal of February 17, 1934, many speakers expressed themselves favourably towards tuberculin. Only one, who admitted that he did not use it, seemed unfavourably impressed.

It is a pity that the present-day teachings are antagonistic to tuberculin, for it is of inestimable value in diagnosis and, in my humble opinion, it is the best weapon we possess in the treatment of tuberculosis, whether pulmonary or surgical.

By the use of tuberculin I have recently been able to diagnose, among others, two cases of renal tuberculosis (later confirmed by finding tubercle bacilli in the urine) and a tuberculous keratitis, cases which had apparently remained undiagnosed for four, twelve and five years respectively.

Surely it is time that the unwarranted prejudice which exists among certain members of the profession was removed and the suffering public was given the benefit of a discovery made over fifty years ago!

Yours, etc.,

W. BRUCE FRY.

British Medical Association House,
137, Macquarie Street,
Sydney,
February 23, 1934.

OCULISTS AND OPTOMETRISTS.

SIR: Apparently when the subject of "Oculists and Optometrists" was before the Hobart Congress very definite reference to optometrists'—or opticians'— charges was made in turn by Dr. Cohen, Dr. Pockley, and others (THE MEDICAL JOURNAL OF AUSTRALIA, February 24, 1934). Permit me to say that in my humble judgement such reference was as much out of place at a medical congress as would have been any reference to or discussion on oculists' fees at an optometrical congress.

I am not concerned with the fees charged by persons individually or collectively, nor do I offer the apologies which any group of persons can always find are necessary for individual members. Optometrists and opticians in the main appear to be a desirable group of citizens carrying on a necessary activity in the community, and in this respect they do not differ from medical practitioners. I suggest, therefore, that it was unjust that the papers on "Oculists and Optometrists" were dealt with at the congress, as they were, especially as the relevant facts stated are incorrect.

It is with reluctance that I direct your attention to the fact that the name of a deceased Sydney optometrist is associated with a bed endowed in perpetuity by his daughter in one of our public hospitals, and also that names of optometrists are to be found in the lists of annual subscribers to hospital funds.

Yours, etc.,

W. I. WENBORN.

2A, Castlereagh Street,
Sydney,
March 5, 1934.

A PROTEST.

SIR: The following paragraph appeared in the Adelaide *Advertiser* of March 1, under a report of the meeting of the Australasian College of Surgeons.

Operations by General Practitioners.

It was not the policy of the college to object to a general practitioner performing an operation himself provided he was quite clear as to the possible result, Sir Henry said. If he were not clear, his conscience ought to lead him to consult an experienced surgeon in order to gain a clear idea of the indications, dangers, and technique of the operation. The doctor was most to be commended who established a correct diagnosis at once, and regardless of other considerations sent his patients for early operation to the place where there was the best prospect of a permanently successful result. He could then consider that he had been the chief cause of the success, and he was certainly more to be congratulated than the man who, unconscious of his limitations, could not resist the temptation of trying to prove his ability as an operator under conditions where the requisites for success were unattainable.

To the average lay person this paragraph is a distinct slur on the operating powers of the general practitioner and will cause him quite a lot of harm.

I am not concerned with the question of whether a general practitioner should operate or not, although, in fairness to the large number of general practitioners, I should say that the average man knows his limitations only too well. I also know a very large number of general practitioners who are remarkably good surgeons.

I am complaining about its publication in a daily paper. Its correct place of discussion is at a meeting of medical men.

To the average reader the paragraph appears as thinly veiled propaganda for increased surgical specialization, and, to say the least of it, is in very bad taste and decidedly unethical.

Yours, etc.,

"G.P."

Adelaide,
March 1, 1934.

Obituary.

MARY JOSEPHINE STACK.

WE regret to announce the death of Dr. Mary Josephine Stack, which occurred at Melbourne on March 11, 1934.

HAROLD SELWYN CAPPER.

WE regret to announce the death of Dr. Harold Selwyn Capper, which occurred at Avoca Beach, Gosford, New South Wales, on March 13, 1934.

Corrigendum.

LEONTIASIS OSSEA.

DR. H. R. SEAR writes that a serious error has appeared in the summary of his presidential address to the Section of X Ray and Radium Therapy, published in the issue of March 3, 1934. He states that he certainly drew an analogy between *leontiasis ossea* and *mélorhéostose*, but adds that this concerned the spread of the bony overgrowth into the soft tissues after rupture of the periosteum. He did not state that leontiasis spread "towards the periphery in a linear fashion, instead of diffusely over the bone".

The words used by Dr. Sear were as follows:

> There is one other dystrophy in which there occurs a deep, very dense, and sclerotic periosteal overgrowth, namely, Léri's *mélorhéostose*, or Junghagen's monomeric eburnous osteosis. In this dystrophy osteosclerotic bone may also appear radiographically to rupture through the enveloping periosteum, leading to the formation of large bony masses in the soft tissues. Röntgenological evidence suggests that these two lesions are closely allied, and possibly they may be due to the same cause.

Later on he wrote:

> The radiographic appearance of the periostitic overgrowth characteristic of *leontiasis ossea* seems to be very similar, if not identical, with that of the dys-

trophy in the long bones described by Léri as *mélorhéostose* and also by Junghagen and Lewin. In the latter dystrophy, as already pointed out, one is impressed by the fact that the osteosclerosis spreads towards the periphery in a linear fashion instead of diffusely over the bone.

We regret that in the summary of Dr. Sear's remarks a false impression was created.

Books Received.

AIDS TO SANITARY SCIENCE AND LAW, by C. F. White, M.B., Ch.B., D.P.H., D.T.M., revised by H. Willoughby, M.R.C.S., L.R.C.P., D.P.H., D.T.M. and H.; Second Edition; 1934. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 328. Price: 4s. 6d. net.

A SHORT HISTORY OF THE EPIDEMIC INFECTIOUS DISEASES, by E. W. Goodall, O.B.E., M.D.; 1934. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 113. Price: 3s. 6d. net.

GENERAL PRACTICE SERIES: RHEUMATISM IN GENERAL PRACTICE, A CLINICAL STUDY, by M. B. Ray, M.D., with foreword by Lord Horder of Ashford, K.C.V.O., M.D., F.R.C.P.; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 414, with six plates. Price: 16s. net.

Diary for the Month.

Mar. 27.—New South Wales Branch, B.M.A.: Council: Election of Office-Bearers and Appointment of Standing Committees.
Mar. 28.—Victorian Branch, B.M.A.: Council.
Mar. 29.—South Australian Branch, B.M.A.: Branch.
April 3.—Tasmanian Branch, B.M.A.: Council.
April 4.—Western Australian Branch, B.M.A.: Council.
April 5.—South Australian Branch, B.M.A.: Council.
April 6.—Queensland Branch, B.M.A.: Branch.
April 10.—Tasmanian Branch, B.M.A.: Branch.
April 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
April 13.—Queensland Branch, B.M.A.: Council.
April 17.—Tasmanian Branch, B.M.A.: Council.
April 17.—New South Wales Branch, B.M.A.: Ethics Committee.
April 18.—Western Australian Branch, B.M.A.: Branch.
April 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
April 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. D. L. Barlow (B.M.A.) has been appointed Bacteriologist in Charge of the Vaccine and Asthma Clinic, Adelaide Hospital, South Australia.

* * *

Dr. E. R. Crisp has been appointed Temporary Honorary Clinical Assistant to the X Ray Department, Adelaide Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv and xv.

Launceston Public Hospital, Launceston, Tasmania: Resident Medical Officer (male).
Parramatta District Hospital, Parramatta, New South Wales: Junior Resident Medical Officer.
Royal Prince Alfred Hospital, Sydney, New South Wales: Honorary Officers.
St. Andrew's Presbyterian Hospital, Melbourne, Victoria: Matron.
The Brisbane and South Coast Hospitals Board, Queensland: Clinical Assistant.
Toowoomba Hospitals Board, Toowoomba, Queensland: Resident Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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